COMPARATIVE STUDY OF ANTERIOR AND INFERIOR WALL MYOCARDIAL INFARCTION

Dissertation submitted in partial fulfillment of regulation for the award of M.D. Degree in General Medicine (Branch I)

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

Chennai

April 2010

DEPARTMENT OF GENERAL MEDICINE

COIMBATORE MEDICAL COLLEGE & HOSPITAL

CERTIFICATE

Certified that this is the bonafide dissertation done by

DR. HIMAL RAJ.M. *and submitted in partial fulfillment of the requirements for the Degree of M.D., General Medicine, Branch I of The Tamilnadu Dr. M.G.R. Medical University, Chennai.*

Date : Professor and Chief of Medical Unit 3

Date : Professor & Head
Department of Medicine

Date : Dean
Coimbatore Medical college

DECLARATION

I solemnly declare that the dissertation titled **"COMPARATIVE STUDY OF ANTERIOR AND INFERIOR WALL MYOCARDIAL INFARCTION "** was done by me from January 2009 to June 2010 under the guidance and supervision of **Professor Dr.S.USHA MD**

This dissertation is submitted to the Tamilnadu Dr. MGR Medical University towards the partial fulfillment of the requirement for the award of MD Degree in General Medicine (Branch I).

Place : Coimbatore **Dr.HIMAL RAJ.M.**

Date :

ACKNOWLEDGEMENT

I express my thanks to our respected **Dean Dr.R.Vimala M.D**., and our medical superintendent **Dr.A.Mathivanan, DCH.M.S,** for permitting me to conduct this study.

I express my deep sense of gratitude to our Head of the Department, **Prof.Dr S.Veerakesari.M.D** and former HOD **Prof. Dr. M. Ramaswamy.M.D**. for their valuable guidance.

I owe a great debt of gratitude towards my Unit Chief **Prof. Dr.S.Usha, M.D**., for her valuable suggestions and guidance to do this study.

I extend my gratitude to Head of Department of Cardiology **Prof. Dr.D.Dharmarajan. M.D.,D.M.**, for his guidance and assistance during the entire study.

I extend thanks to **Prof Dr.Nedumaran, M.D., D.M**., the Professor of therapeutics and **Prof Dr Raveendran M.D** for their overwhelming enthusiasm and guidance during the entire duration of study.

I whole heartedly thank my unit assistant professors, **Dr.B.Vetriveeran,M.D.,Dr.R.Balamurugan,M.D., Dr.V.Ushapadmini, M.D**., for their able guidance.

I am indebted to all my patients and their family members for their sincere cooperation, without whom, this study would not have been successful.

CONTENTS

Introduction

INTRODUCTION

Myocardial infarction or necrosis of myocardial cells is one of the commonest diagnoses in hospitalised patients. It is one of the three main killers of mankind , ie , Myocardial infarction, cerebrovascular accidents and cancer . Myocardial Infarction is a serious complication of Atherosclerotic Coronary Heart Disease[1]. In 2001, it was estimated that worldwide, ischemic heart disease was responsible for 11.8 percent of all deaths (5.7 million) in low-income countries and 17.3 percent (1.36 million) of all deaths in high-income countries. One-third of Acute myocardial infarction are caused by an acute ST-segment elevation myocardial infarction (STEMI)[2]. The incidence of AMI[3] has declined over the past two decades from 244 per 100,000 population in 1975 to 184 per 100,000 population in 1995[4].

Coronary Heart Disease was first described by William Herberden in 1768. In 1932 , unipolar leads were discovered by Wilson and the establishment of coronary care units by Day and Brown has lead to the world wide proliferation of coronary care units. The current knowledge of pathophysiology of acute myocardial infarction started with the autopsy description of Dr. James Herrick from Chicago in 1912 who concluded that acute myocardial infarction results from thrombotic occlusion of coronary artery and prophesized that the hope of salvaging the muscle,

lay in restoration of blood flow. In the landmark paper by Herrick[5], he wrote, “The clinical manifestations of coronary obstruction will evidently vary greatly depending on the size, location and number of vessels occluded. The symptoms and end-result must also be influenced by blood pressure, by the condition of the myocardium not immediately affected by the obstruction, and by the ability of the remaining vessels to properly carry on their work, as determined by their health or disease”

Myocardial Infarction[6] is an acute cardiac disability arising from reduction or arrest of blood supply to the myocardium due to atherosclerotic or non-atherosclerotic lesions of coronary arteries. Virtually all acute infarcts are caused by thrombosis developing in a culprit vessel with ruptured atherosclerotic plaque. Usually coronary artery occlusion is associated with infarction of myocardium, though post-mortem examination of cases of sudden deaths reveals evidence in only 20% of cases, the remaining 80% not showing any change.

The incidence of myocardial infarction[7] increases with both sex and social class. It is three to four times more frequent among men than women . The popular concept that coronary artery disease is the scourge of the managerial and industrial class appears to be wrong . Recent surveys suggest that myocardial infarction is substantially more common among manual and skilled workers.This observation is only partially explained in terms of classical risk factors. Approximately 40 –

50 % of patients experiencing a heart attack die within 20 days of the onset . Elderly patients invariably fare less well than the young and middle aged . Half the deaths occur between 1 and 2 hours of the onset of symptoms . Many patients therefore fail to reach the hospital , succumbing to the potentially correctable ventricular fibrillation. In contrast , a person surviving long enough to gain admission to hospital is more likely to die as a result of pump failure , intractable left ventricular failure or cardiogenic shock for which even the best coronary care unit can do little.

Mortality from STEMI has declined steadily in several population groups since 1960[8,9]. This drop in mortality appears to result from a fall in the incidence of STEMI (replaced in part by an increase in the rate of unstable angina/non-ST-segment elevation MI[10]) and a fall in the case fatality rate once STEMI has occurred[11].Several phases in the management of patients have contributed to the decline in mortality from STEMI[12]. The "clinical observation phase" of coronary care consumed the first half of the 20th century and focused on a detailed recording of physical and laboratory findings, with little active treatment for the infarction[13]. The "coronary care unit phase" began in the mid-1960s and was notable for detailed analysis and vigorous management of cardiac arrhythmias. The "high-technology phase" was ushered in by the introduction of the pulmonary artery balloon floatation catheter,

setting the stage for bedside hemodynamic monitoring and more precise hemodynamic management. The modern “reperfusion era” of coronary care was introduced by intracoronary and then intravenous fibrinolysis, increased use of aspirin, and development of primary percutaneous coronary intervention (PCI)

Accurate diagnosis is mandatory because mistaken diagnosis can be disastrous to the social, economical and family life of the patient. At one end of the spectrum is the danger of missing a potentially lethal illness, on the other hand mistaken diagnosis results in severe cardiac neurosis which is even more difficult to treat than the original disease itself.

Aim of the study

AIM OF THE STUDY

The aims of the study are :

1. To find out the overall incidence of anterior and inferior wall myocardial infarction.

2. To compare the incidence and mortality rate of anterior and inferior wall myocardial infarction in relation to age , sex and occupation.

3. To compare the effects of various risk factors on anterior and inferior wall myocardial infarction.

4. To compare the incidence and mortality rate of anterior and inferior wall myocardial infarction , with reference to Killip's classification of left ventricular failure .

5. To compare the incidence of arrhythmias in anterior and inferior wall myocardial infarction and mortality due to arrhythmias .

Review of Literature

REVIEW OF LITERATURE

A. ANATOMY OF CORONARY CIRCULATION[14] (Fig 1)

Right coronary artery (RCA)

The **right coronary artery** originates from the right aortic sinus of the ascending aorta. During this course, several branches arise from the main stem of the vessel:

- an early **atrial branch** passes in the groove between the right auricle and ascending aorta, and gives off the **sinu-atrial nodal branch** to supply the sinu-atrial node;
- a **right marginal branch** is given off and continues along this border toward the apex of the heart;
- a small branch to the atrioventricular node
- before giving off its final major branch, the **posterior interventricular branch**,

The right coronary artery supplies the right atrium and right ventricle, the sinu-atrial and atrioventricular nodes, the interatrial septum, a portion of the left atrium, the posteroinferior one-third of the interventricular septum, and a portion of the posterior part of the left ventricle

Left coronary artery (LCA)

The **left coronary artery** originates from the left aortic sinus of the ascending aorta. While still posterior to the pulmonary trunk, the artery divides into its two terminal branches, the anterior interventricular and the circumflex .

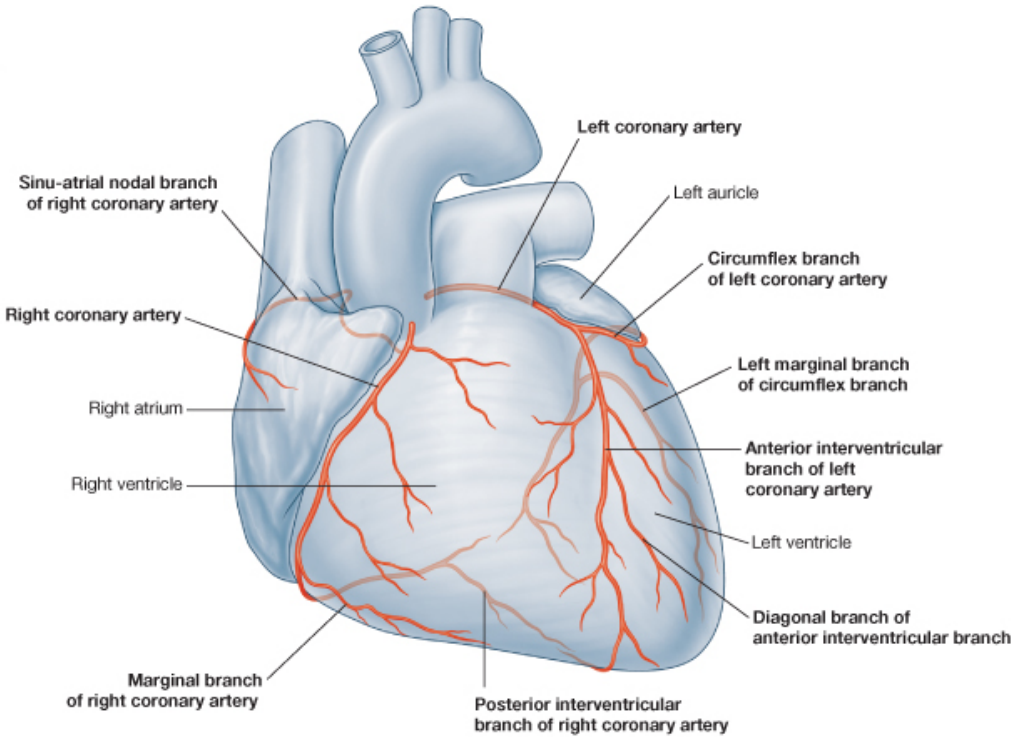
- the **anterior interventricular branch (left anterior descending artery-LAD)**, which continues around the left side of the pulmonary trunk and descends obliquely toward the apex of the heart in the anterior interventricular sulcus.
- the **circumflex branch**, ends before reaching the posterior interventricular sulcus-
- a large branch, the **left marginal artery**,

  The distribution pattern of the left coronary artery enables it to supply most of the left atrium and left ventricle, and most of the interventricular septum, including the atrioventricular bundle and its branches.

In 85% of patients, the right coronary artery which gives rise to posterior descending artery supplies the entire right ventricle and large a part of the posterior wall of the left ventricle. This is referred to as right dominant circulation.

In 8% of patients the left coronary artery supplies entire left ventricle, interventricular septum and a portion of right ventricle. This is referred to as left dominant circulation .

Fig 1



B.DEFINITION OF MYOCARDIAL INFARCTION

Epidemiological reports from the World Heath Organization and American Heart Association beginning in the late 1950s required the presence of at least two of the following for the diagnosis of myocardial infarction: characteristic symptoms, electrocardiographic changes, and a typical rise and fall in biochemical markers[15].

Revised Definition of Myocardial Infarction[16](Table 1)

Table 1

Criteria for Acute, Evolving, or Recent MI
Either of the following criteria satisfies the diagnosis for acute, evolving, or recent MI:
1. Typical rise and/or fall of biochemical markers of myocardial necrosis (CK-MB or troponin)with at least one of the following: **a)** Ischemic symptoms **b)** Development of pathological Q waves in the ECG **c)** ECG changes indicative of ischemia (ST segment elevation or depression) **d)** Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality **2.** Pathological findings of an acute myocardial infarction
Criteria for Healing or Healed Myocardial Infarction
Any one of the following criteria satisfies the diagnosis for healing or healed myocardial infarction:
1. Development of new pathological Q waves in serial ECGs. The patient may or may not remember previous symptoms.

Biochemical markers of myocardial necrosis may have normalized depending on the length of time that has passed since the infarction developed.

2. Pathological findings of a healed or healing infarction

Following disruption of a vulnerable plaque, patients experience ischemic discomfort resulting from a reduction of flow through the affected epicardial coronary artery. Of patients with ST-segment elevation, the majority ultimately develop a Q-wave on the ECG (QwMI), while a minority do not develop Q-wave and in older literature were said to have sustained a non-Q-wave MI (NQMI). Patients who present without ST-segment elevation are suffering from either unstable angina or a non-ST-segment elevation MI (NSTEMI) , a distinction that is ultimately made on the presence or absence of a serum cardiac marker such as CKMB or a cardiac troponin detected in the blood. The majority of patients presenting with NSTEMI do not develop a Q-wave on the ECG; a minority develop a QwMI[17] .(Fig 2)

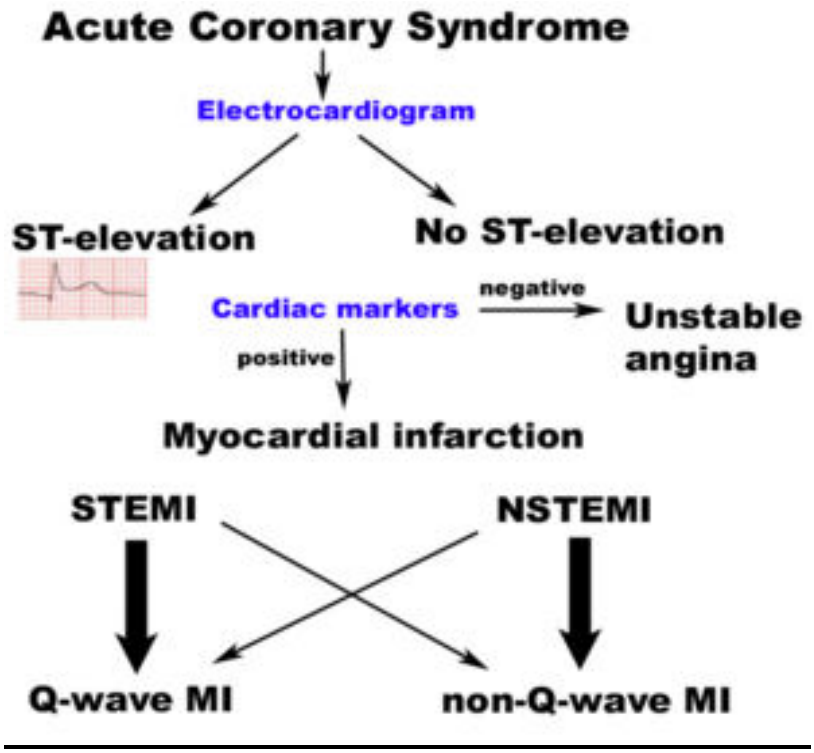


Fig 2. Scheme for diagnosis of Myocardial Infarction

C.AETIOPATHOGENESIS OF MYOCARDIAL INFARCTION

1.ATHEROSCLEROSIS[18,19,20]

Atherosclerosis is characterized by formation of atheroma or fibro fatty plaque which consists of a raised focal plaque within the intima, having a core of lipid and a covering of fibrous cap. (**Fig 3**) Atherosclerosis[21,22,23] is the single most important etiological factor for coronary heart disease. The search for the cause and pathogenesis of atherosclerosis[24,25,26] has become an insistent "golden grail". The acceptable hypotheses are:

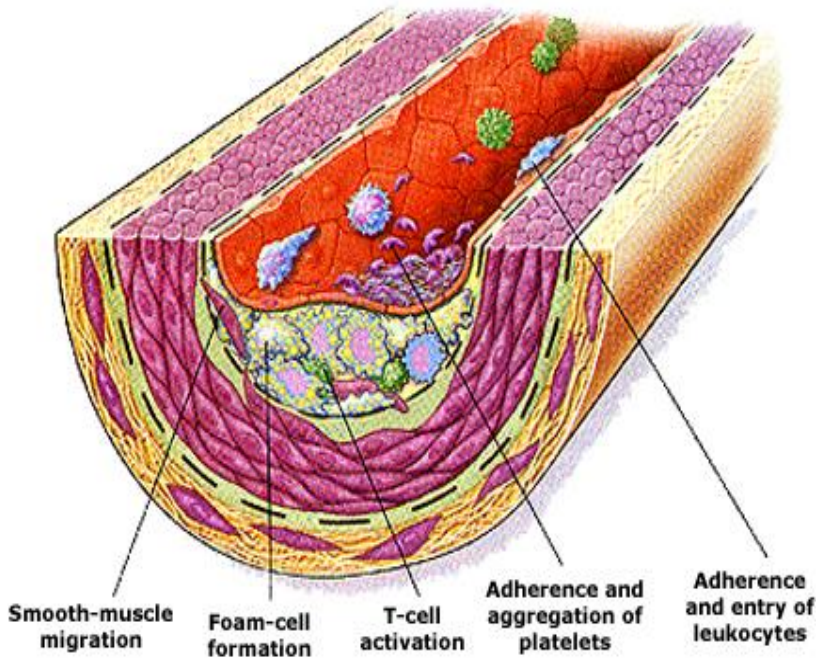


Fig 3. Fatty-streak formation in atherosclerosis

A) LIPID INSUDATION OR INFILTRATION HYPOTHESIS:

This is the modified "imbibition hypothesis"[27]termed by Virchow in 1856 which stated that cellular proliferation in intima was a form of low grade inflammation as a reaction to increased infiltration of plasma protein and lipids[28] from the blood.

B) ENCRUSTATION OR THROMBOGENIC HYPOTHESIS:

This theory ascribed to Rokitansky, postulated that small thrombi composed of platelet, fibrin and leukocytes collected over foci of endothelial injury organized and their gradual growth resulted in plaque formation[29,30]

C) REACTION TO INJURY HYPOTHESIS:

This widely accepted theory formulated by Ross[31] and Glomset in 1976 and modified in 1986[32,33] states that the lesions of atherosclerosis are initiated as a response to some form of injury to arterial endothelium. Endothelial injury leads to attachment of monocytes and platelets, proliferation of smooth muscle cells in the arterial intima and deposition of intracellular and extracellular lipids.

D INFLAMMATION THEORY

In his most recent review of the pathogenesis of atherosclerosis , Ross[34] continues to state the importance of endothelial dysfunction in the origin of atherosclerosis but also highlights the role played by inflammation at every step of the pathogenesis of atherosclerosis.

1.NON-ATHEROSCLEROSIS [35,36]

Non-atherosclerotic[37,38,39] causes of Myocardial Infarction[,40,41,42] are

1. Congenital anomalies like single coronary artery, atresia of coronary ostium, myocardial bridges, coronary AV fistula etc.
2. Dissection of coronary artery or aorta.
3. Embolic phenomena from prosthetic valves, infective endocarditis, tumors, calcium, paradoxical embolus, etc.
4. Traumatic injury or spasm of coronary artery.

5. Coronary arteritis due to Takayasu Disease, Polyarteritis Nodosa, SLE, Syphilis, Kawasaki Disease
6. Metabolic disorders like Mucopolysaccharidoses, Homocystinuria, Fabry's Disease and Amyloidosis.
7. Substance abuse like cocaine, amphetamine.
8. Myocardial oxygen demand-supply disproportion due to aortic stenosis, systemic hypotension, carbon monoxide poisoning, thyrotoxicosis.
9. Intimal proliferation due to irradiation, cardiac transplantation, fibro muscular hyperplasia.
10. Miscellaneous cause like HOCM, hypercoagulable states, diabetes mellitus.

3. RISK FACTORS FOR MYOCARDIAL INFARCTION [43,44,45] (TABLE 1)

The term risk is widely used to describe those characteristically found in healthy individuals that relate to the subsequent appearance of Ischaemic Heart Disease[46,47,48]. The risk of Ischaemic Heart Disease[49,50,51] is determined by the aggregation of individual factors.

Table 1: RISK FACTORS FOR CORONARY HEART DISEASE

A. FIXED
1. Age 2. Male sex 3. Family History
B. MODIFIABLE
1. Smoking 2. Hypertension 3. Lipid Disorders 4. Diabetes Mellitus 5.Haemostatic variables 6. Sedentary Life Style 7. Obesity 8. Mental Stress 9. Personality 10. Oral Contraceptive Pills 11. Hyperhomocysteinemia 12. Inflammation

1. AGE:

Age[52,53] is a definite unmodifiable risk factor. Atherosclerosis develops progressively as age advances. Men acquire an independent risk factor at age 45, Women acquire an independent risk factor at age 55 . Atherosclerosisis rarely present in early childhood, except in familial hyperlipidemia, but it is often detectable in postmortem

specimens of young age between 15-30 years. Atherosclerosis is universal in elderly.

2. SEX:

Men are more affected than premenopausal women. However after menopause the incidence of atheroma rises in women[54,55,56]. This suggests that oestrogen probably plays a part in preventing or delaying atherosclerosis. There is also a fall in HDL levels in postmenopausal women which may also contribute.

3. FAMILY HISTORY:

Coronary artery disease runs in families. This may be due to genetic factors or the effects of a shared environment (similar diet, smoking habits etc,). A positive family history[57,58] is generally accepted to refer to those patients in whom a first degree relative of the patient has developed Ischaemic Heart Disease before the age of 50 years.

4. SMOKING:[59,60]

Tobacco is probably the most important avoidable cause of coronary disease. The incidence of Ischaemic Heart Disease is 3-5 times higher in smokers who smoke 20 cigarettes per day compared to non-smokers. There is a strong, consistent and dose linked relationship between cigarette smoking[61,62] and Ischaemic heart disease. The incidence of sudden death is also higher in smokers. Smoking decreases

HDL cholesterol levels. It accelerates atherosclerosis, plaque instability, increases the risk of thrombosis, myocardial infarction and death.

5. HYPERTENSION[63,64,65]

The incidence of coronary heart disease increases as blood pressure rises and the excess risk is related to both systolic and diastolic blood pressure[66,67]. In the Framingham study, the incidence of Coronary Artery Disease in middle aged persons with blood pressure exceeding 160/95mmHg was 5 times more than that in normotensive men. For each 10mmHg increase in Diastolic Blood Pressure there is 37% increase in risk of Coronary Heart Disease. Antihypertensive drugs have shown to reduce coronary mortality particularly by interruption of renin angiotensin system.

6. LIPID DISORDERS[68,69,70]:

A wealth of evidence from epidemiological, clinical and experimental studies has established the association between hyperlipidemia[71] and atherosclerosis. Hypercholesterolemia is clearly a risk factor.

Of the lipoprotein, it is the low density lipoprotein (LDL) which is most atherogenic. VLDL is comparatively less atherogenic. HDL offers a protective effect and helps in removing cholesterol from the

arterial wall. The ratio of LDL/HDL is a common way to assess atherogenicity of hyperlipidemia. A ratio of more than 4.5 is supposed to be atherogenic. A minor increase of 1mg/dl in HDL-4 Cholesterol produces a 2-4% decrease in the risk of developing Acute Myocardial Infarction.

The LDL cholesterol profiles are categorized as phenotypic pattern A, B and C. Patients with pattern B have mostly small, dense LDL particles. Small, dense LDL particles have been causally linked to an increased risk of coronary artery disease independent of total LDL cholesterol levels. It has been suggested that small dense LDL particles are more atherogenic because of greater retention in the arterial wall and increased susceptibility to oxidation.

There is increasing evidence that hypertriglyceridemia is independently linked with coronary atheroma even after adjustment for HDL levels

7. DIABETES MELLITUS[72,73]:

Diabetes Mellitus[74] is a coronary heart disease risk equivalent. The abnormal lipid profile with insulin resistance known as Diabetic Dyslipidemia (small dense LDL, low HDL, elevated triglyceride) account for part of elevated cardiovascular risk. Diabetes increases the frequency of atherosclerosis. It is likely that post prandial hyperglycemia may be more important in the development of coronary

artery disease than fasting hyperglycemia. Diabetic women are 5-6 times more prone for coronary heart disease compared to men.

8. HAEMOSTATIC FACTORS[75,76]

High levels of fibrinogen and factor 7 are associated with increased risk of myocardial infarction. Polymorphism of factor 7 gene may increase the risk of Myocardial Infarction. The stability of an arterial thrombus depends on the balance between fibrinolytic factors like plasmin and inhibitors of the fibrinolytic system such as Plasminogen Activator Inhibitor (PAI) 1. However, the levels of tissue plasminogen activator and PAI-1 in plasma have not proven to add information beyond the lipid profile for assessment of cardiovascular risk.

Apolipoprotein (a) has structural homology with plasminogen with which it is able to compete for cell surface binding. By displacing plasminogen, apolipoprotein (a)[77] is able to inhibit cell surface mediated endogenous fibrinolysis by reducing the formation of Plasminogen Activator from Plasminogen. Plasma level of Lp (a) is largely genetically determined. Lp (a) levels do not potently predict risk in the population at large.

9. SEDENTARY LIFE STYLE[78]:

The recent National Institute of Health Consensus Panel on Physical Activity and Cardiovascular Health established a goal of atleast

30 min of moderate intensity physical activity on a daily basis. Exercise increases HDL cholesterol, lowers blood pressure, reduces blood clotting and promote collateral vessel development.

10. OBESITY:

Obesity[79,80], particularly the male pattern of centripetal or visceral fat accumulation is probably an independent risk factor although it can be associated with hypertension, diabetes mellitus and physical inactivity.

11. MENTAL STRESS AND PERSONALITY:

Stress is associated with increased catecholamine levels and high blood pressure thereby increasing the risk of coronary heart disease.

Type A individuals who are ambitious, aggressive, impatient, competitive, always in a hurry and often frustrated are more prone to Ischaemic heart disease.

12. ORAL CONTRACEPTIVE PILLS:

Oral contraceptives disturb the clotting mechanism through an increased inactivity of factor 7 and 10. It also increases platelet sensitivity to ADP and hence to platelet adhesiveness. Oestrogen retards atherosclerosis but accelerates thrombogenesis resulting in myocardial infarction in the rare susceptible person.

13. HYPERHOMOCYSTEINEMIA:

A large body of literature suggests a relationship between hyperhomocysteinemia and coronary events. Several mutations in the enzymes involved in homocysteine[81] accumulation correlate with thrombosis and in some studies, coronary risk. Measurement of homocysteine levels should be reserved for individuals with atherosclerosis at a young age or out of proportion to established risk factors.

14. INFLAMMATION[82]:

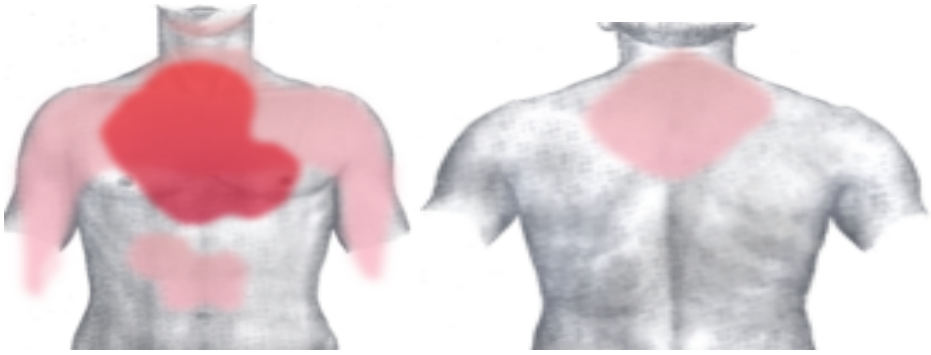
An accumulation of clinical evidence shows that markers of inflammation correlate with coronary risk. Inflammatory cells in the plaque may contribute to plaque destabilization by producing matrix degrading metalloproteinases and by inducing smooth muscle cell apoptosis. Inflammatory cells also contribute to plaque thrombogenicity by releasing tissue factor- a procoagulant protein that activates the clotting cascade resulting in thrombin generation which leads to platelet aggregation and fibrin deposition. Inflammation in atherosclerotic plaque may be incited by a number of factors, which include oxidized LDL, cigarette smoking and possibly infectious agents. Variation of plasma levels of CRP can prospectively predict risk of myocardial infarction. CRP[83] levels also correlate with outcome of patients with acute coronary syndrome. Elevated levels of CRP may merely reflect

ongoing inflammation rather than a direct etiologic role of CRP in the coronary artery disease.

D. CLINICAL FEATURES[84] (Fig 4)

Retrosternal chest discomfort is the cardinal symptom of myocardial infarction, but breathlessness, vomiting, giddiness, syncope are common features. Patient is anxious and experiences the fear of impending death. Syncope is usually due to arrhythmia or profound hypotension. Vomiting and

Fig 4. Rough diagram of pain zones in myocardial infarction (dark red = most typical area, light red = other possible areas, view of the chest).



sinus bradycardia are often due to vagal stimulation and are particularly common in patients with inferior wall myocardial infarction. Many deaths occur within the first hour. Development of cardiac failure reflects the extent of myocardial damage and is the major cause of death in those who survive the first few hours of infarction. On examination signs of sympathetic activity like sweating, tachycardia may be present. Vomiting and bradycardia may also be present. Signs of impaired myocardial

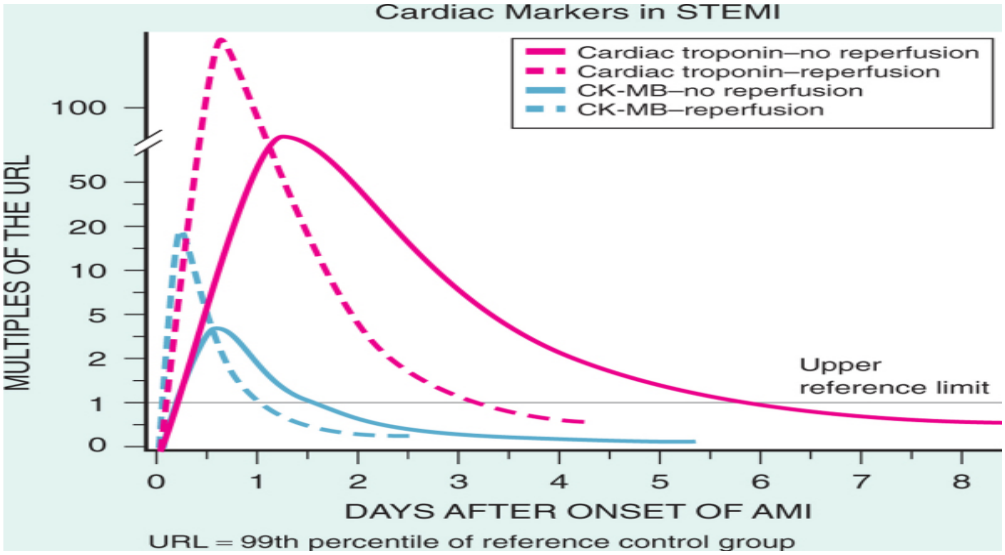
function like hypotension, oliguria, cold peripheries, raised JVP, muffled heart sounds, S3, basal crepitations, pansystolic murmurs may also be present. Pericardial rub may be present.

E.INVESTIGATIONS

1. LABORATORY FINDINGS:

Myoglobin levels are the earliest to rise. Creatine Kinase starts to rise at 4 hours, peaks at about 12 hours falls to normal levels within 48-72 hours [85,86]. The most sensitive markers of myocardial cell damage are the Cardiac Troponins T and I which are released within 4-6 hours and remain elevated for upto 2 weeks. Myoglobin levels peak at 6 hours and returns to normal at 24 hours. Aspartate transaminase starts to rise about 12 hours after infarction and reaches peak on the first or second day returning to normal within 3 or 4 days. Lactate dehydrogenase peaks at 3-4 days remains elevated for upto 10 days.

Fig 5

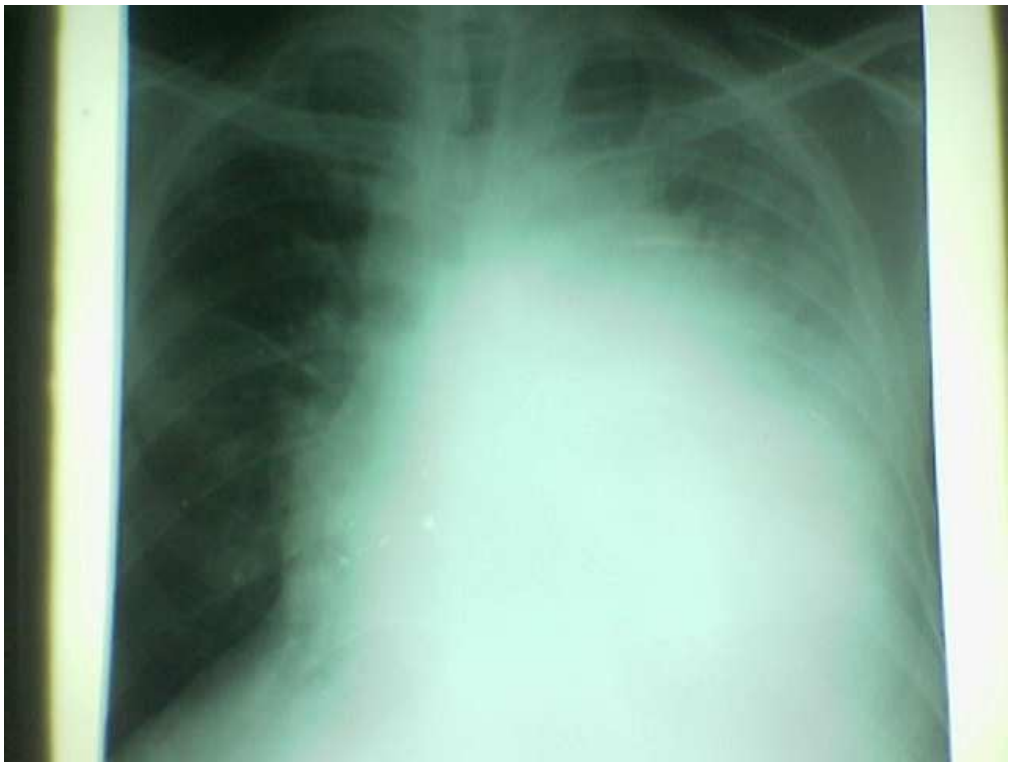


Lipid profile (cholesterol – total, LDL & triglycerides) may be raised. Leukocytosis is usual, reaching a peak on the first day. The ESR becomes raised.

2. CHEST RADIOGRAPHY (Fig 6):

Chest X-ray is important since it may show the consequences of ischaemic heart disease i.e. cardiac enlargement, ventricular aneurysm, signs of heart failure and pericardial effusion. These signs can support the diagnosis of ischaemic heart disease and are important in assessing degree of cardiac damage.

Fig 6 CXR showing pericardial effusion



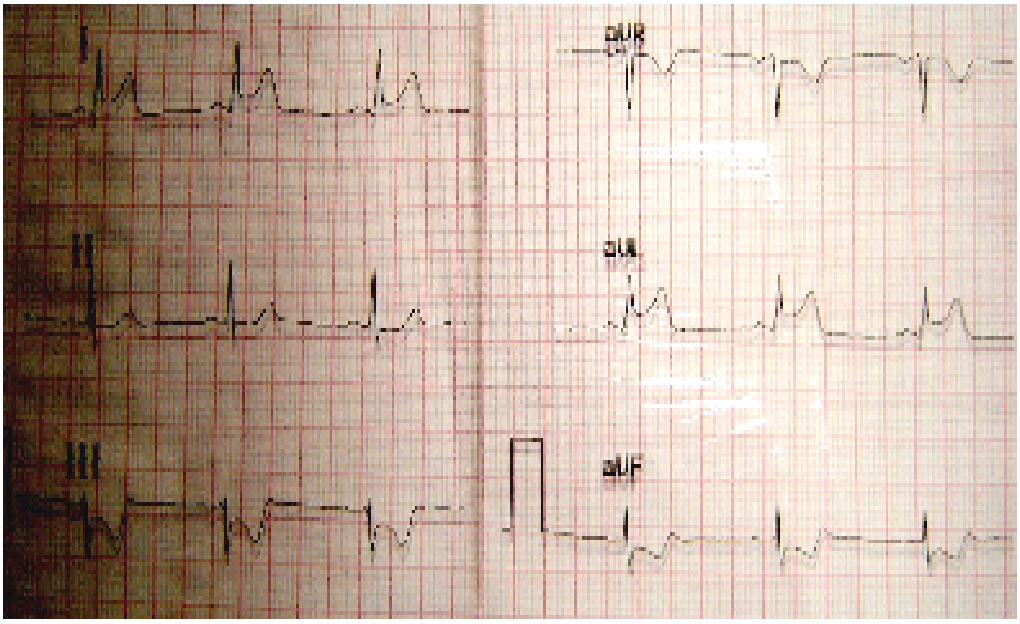
3. ELECTROCARDIOGRAPHY[87,88,89](FIG 7,8,9)(Table 2)

The ECG[90] is usually a sensitive and specific way of confirming the diagnosis; however it may be difficult to interpret if there is bundle branch block or evidence of previous myocardial infarction. Occasionally the initial ECG is normal and the diagnostic changes appear a few hours later. The earliest ECG change is tall and widened T waves followed by ST elevation. Later on there is diminution in the size of R wave and in full thickness infarction a Q wave begins to develop. Subsequently T wave becomes inverted.

Table 2

ANATOMICALZONE	ECG ZONE
INFERIOR WALL	LII,III,Avf
ANTERO SEPTALWALL	V1-V4
EXTENSIVE ANTERIOR WALL	LI,AVL,V1-V6
ANTERO LATERAL WALL	LI,AVL,V5-V6
INFERO LATERAL WALL	LII,LIII,AVF,AVL,V5-V6

Fig 7. ECG showing Anterolateral wall Myocardial Infarction



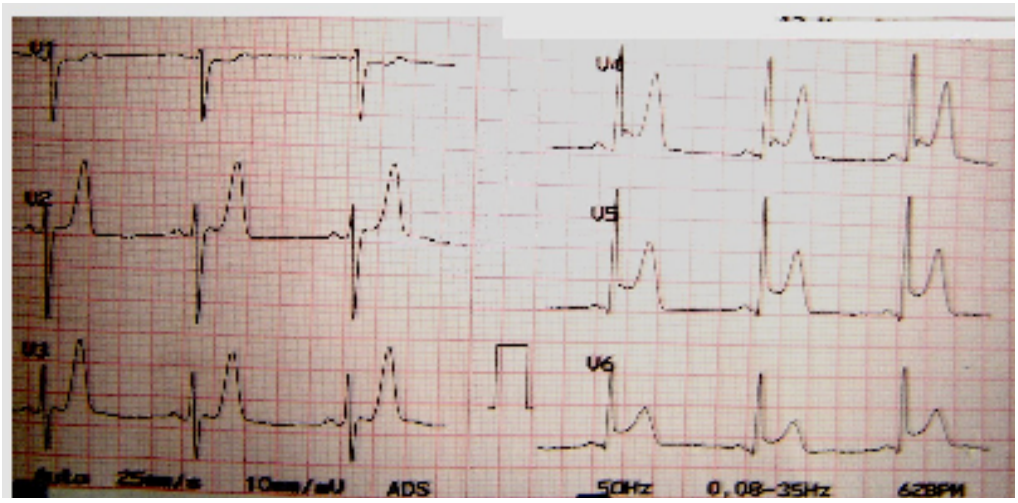
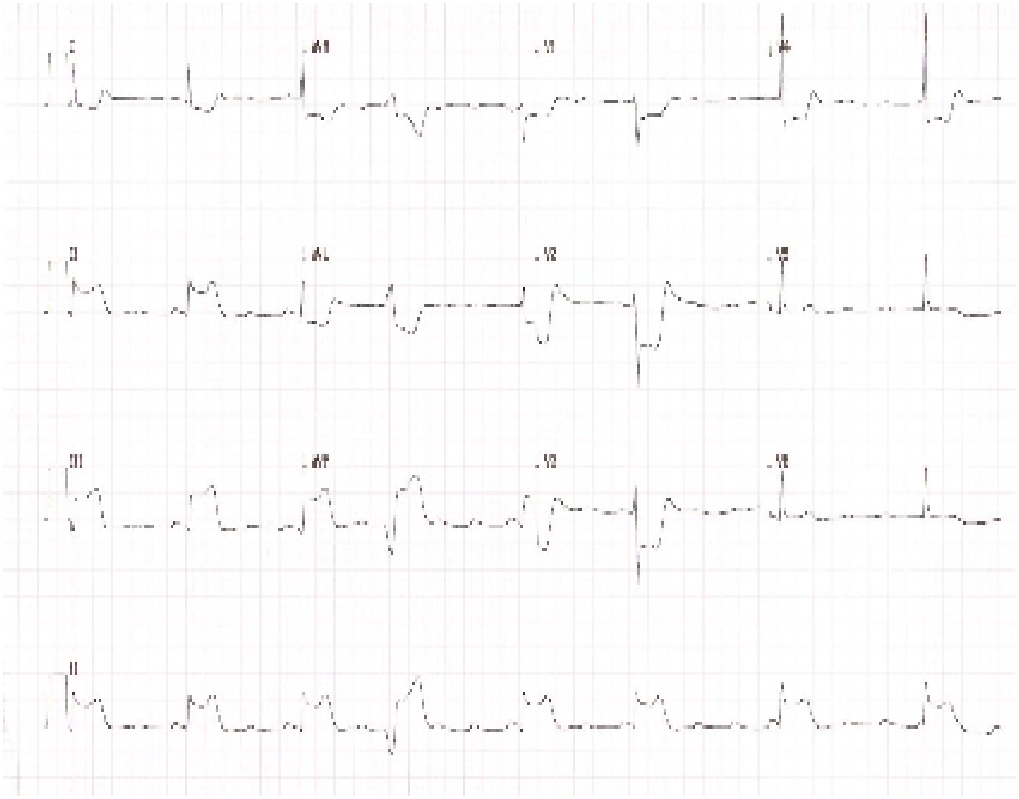


Fig 8. ECG showing inferior wall MI



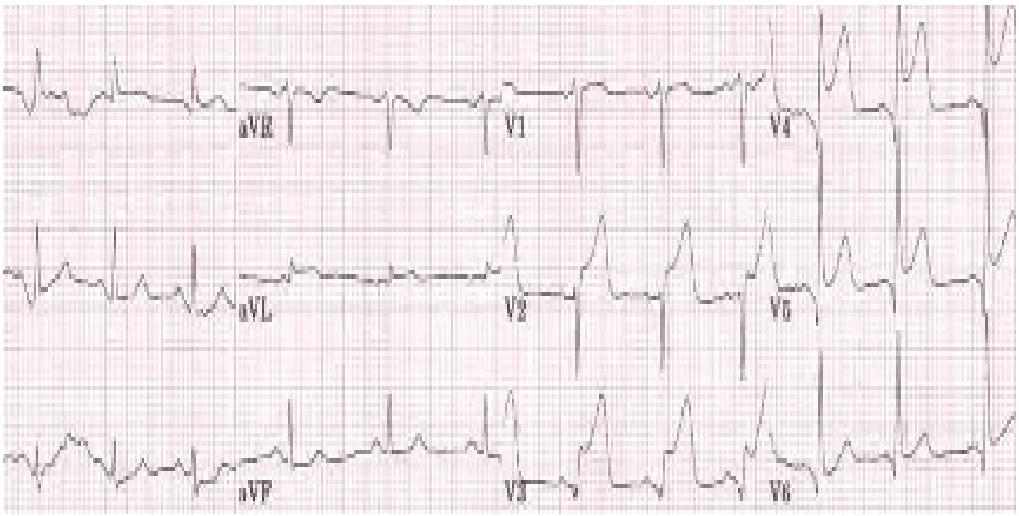


Fig 9.Anterior wall MI

When there has been anteroseptal infarction, abnormalities are found in one or more leads from V1 to V4, while Anterolateral Infarction produces abnormalities in V4-V6, aVL and in lead I. Inferior Wall Infarction is best shown in lead II, III and aVF, while at the same time leads I, aVL and the anterior chest leads may show reciprocal changes of ST depression. Infarction of the posterior wall of the left ventricle is not recorded in the standard leads by ST elevation or Q waves, but the reciprocal changes of ST depression and a tall R wave may be seen in leads V1-V3. Right ventricular infarction should be strongly suspected if, in the clinical setting of acute inferior wall myocardial infarction, there is ST elevation of 1mm or more in lead V1, V4R or any of the extra right precordial leads V4R-V6R.

4. ECHOCARDIOGRAPHY (Fig 10):

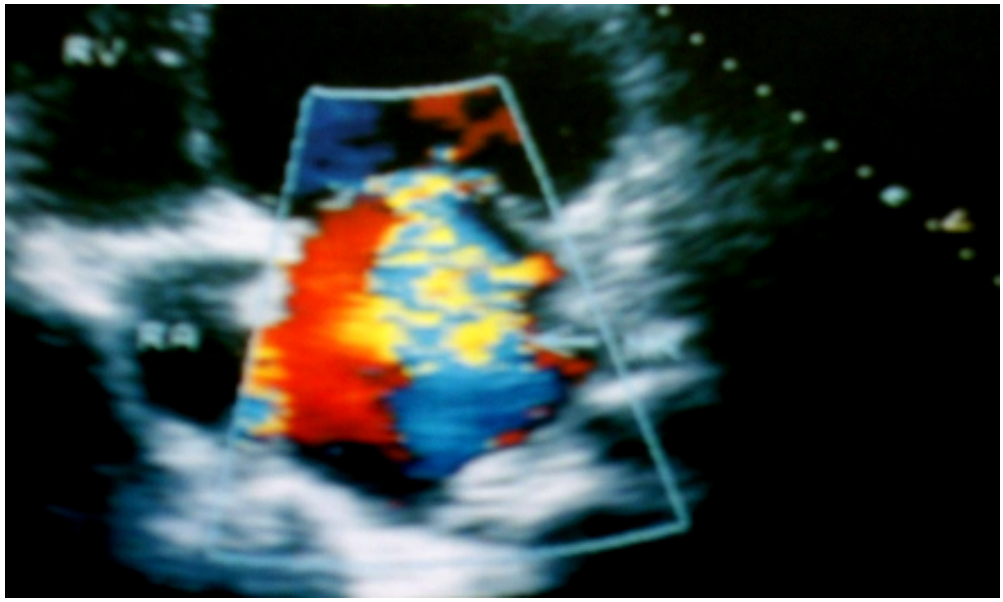
Two dimensional echocardiography [91]can be done to assess the cardiac chamber size, regional wall motion abnormalities, left ventricular hypertrophy, valve leaflet thickness and mobility, valve calcification, appearance of subvalvular and supravalvular structures, pericardial effusion, intracardiac masses and great vessels.

Doppler echocardiography is done to assess valve regurgitation, valve stenosis, valve area, valve gradients, intracardiac pressures, intracardiac shunts and ventricular diastolic filling.

Transesophageal echocardiography is used to assess aortic disease, infective endocarditis, to find out source of embolism, abnormalities of mitral prostheses etc.

Stress echocardiography is done to find out new regional wall motion abnormalities, declining ejection fraction and increase in end systolic volume which are indicators of myocardial ischemia.

Fig 10. ECHO SHOWING ACUTE MITRAL REGURGITATION



5. RADIONUCLIDE SCANNING:

A radionuclide ventriculogram can be used to assess left ventricular function. Infarct 'avid' scanning is possible because some isotopes (e.g. Technetium) are taken up by freshly infarcted myocardium. This may help to establish the diagnosis in some patients who present

after a cardiac arrest when it is sometimes difficult to interpret any ECG and enzyme changes.

F. COMPLICATIONS OF MYOCARDIAL INFARCTION

1. ARRHYTHMIAS[92,93]:

A. TACHYARRYTHMIAS:

a. Premature ventricular complexes:

The commonest arrhythmia is premature ventricular complexes. This can be suppressed with IV lignocaine 100mg given as bolus. To prevent recurrence , lignocaine infusion of 500-1000mg in 500ml of 5% dextrose is administered at a rate of 1-2mg/min. premature ventricular complexes can be forerunners of life threatening ventricular tachycardia or ventricular fibrillation.

b. Ventricular Tachycardia:

It needs immediate attention and should be treated by IV lignocaine. In case the drug is ineffective, DC shock of 150-200 joules will be effective in majority of the cases. Repeated attacks of ventricular tachycardia can be prevented by IV infusion of lignocaine, amiodarone or mexilitene.

c. Ventricular Fibrillation:

The patient is pulseless and will have features of cardiac arrest. Immediate thump on the chest and an external cardiac massage is

required. Defibrillation should be one by DC shock of 200-400 joules. Patients with repeated episodes may benefit from intravenous bretylium, amiodarone, mexilitene or lignocaine.

d. Supraventricular Arrhythmias

Atrial premature beats do not need specific treatment. However supraventricular arrhythmia, atrial flutter and atrial fibrillation require treatment with digoxin or verapamil.

e. Accelerated Ventricular or Junctional Rhythm:

Normally pace maker cells in the AV junctional and ventricular myocardium have a rate of 40-60/min. however in the settings of myocardial infarction, especially in acute inferior wall myocardial infarction, the rate of these pacemakers increases to about 80-120/min. Such a rhythm does not require specific treatment and is normally self limiting. If hemodynamic compromise occurs IV atropine (1.2mg) suppresses it by increasing the sinus rate.

B. BRADYARRHYTHMIAS AND CONDUCTION DISTURBANCES:

a. Sinus Node Dysfunction:

It may present as sinus bradycardia, sinus arrest or sinoatrial block usually due to vagal stimulation or in the settings of inferior wall infarction due to sinus node ischaemia. Symptoms of profound hypotension and shock may occur and occasionally asystole and cardiac

arrest. Immediate treatment consists of IV atropine (1.2mg). If atropine is ineffective or this problem is persistent or recurring temporary pace making is necessary.

b. AV Nodal Block:

It usually occurs with inferior wall myocardial infarction as first degree AV block, Wenkebach's block or complete AV block. These blocks are usually transient and respond to IV atropine. If giddiness, hypotension or other evidence of hemodynamic compromise occurs or if the ventricular rate is less than 50/min temporary pacing may be required.

c. Distal Conduction Disturbances:

The distal conduction system consists of the right bundle and the anterior and posterior fascicles of the left bundle. Since the major blood supply to this part of the conduction system comes from the left coronary artery, conduction defects in the bundle branches are common in anterior wall infarction. Block of one of the fascicles of the left bundle does not have an ominous prognosis. However acute right or left bundle branch block or bifascicular block carries an ominous prognosis. Block in the three fascicles results in complete heart block with an unstable ventricular escape rhythm at a rate of 20-40 beats per minute. Clinical manifestations consists of syncopal attacks, hypotension and may lead to cardiac arrest. Temporary pacemaker insertion is necessary.

Temporary pacing is also indicated in patients who develop bifascicular blocks since this can be a precursor of trifascicular block. Patients with bifascicular block who develop trifascicular block may need permanent pacemaker implantation.

2. ISCHAEMIA:

Post infarction angina occurs in upto 50% of patients. This is due to residual stenosis in infarct related vessel despite successful thrombolysis.

3. ACUTE CIRCULATORY FAILURE:

Hemodynamic evidence of left ventricular dysfunction appears when contraction is seriously impaired in 20-25% of the left ventricle. Infarction of more than 40% of left ventricle results in cardiogenic shock which carries a bad prognosis.

4. PERICARDITIS:

This may occur at any stage but is particularly common on the second and third day. The patient may recognize a different pain that is positional and worsens on inspiration. Dressler's Syndrome may occur between 2 weeks and 3 months after acute myocardial infarction and has an autoimmune basis often accompanied by pleural and pericardial effusions, fever and raised ESR. Treatment requires the use of steroids.

5. MECHANICAL COMPLICATIONS:

a. Mitral regurgitation[94]*:*

It is due to ischaemia or rupture of papillary muscle and is recognized by the presence of systolic murmur at the apex. If trivial, it is of no hemodynamic significance. However severe mitral regurgitation can induce life threatening left ventricular failure and cardiogenic shock and may warrant urgent coronary angiography followed by coronary bypass surgery and mitral valve replacement.

b. Ventricular septal defect:

It is a defect due to rupture[95] of infarcted interventricular septum and is recognized by the presence of pansystolic murmur at the left sternal border. Diagnosis is possible by Echo-Doppler studies. It produces severe left heart failure and needs immediate surgical intervention.

c. Cardiac Rupture[96]*:*

It is a serious complication which results in cardiogenic shock and almost 100% mortality. Emergency treatment by pericardial tapping may prove life saving. Rare cases have been saved by emergency surgery.

6. OTHER COMPLICATIONS[97]:

a. Left Ventricular Aneurysm:

The infarcted segments are dilated and show paradoxical movement and compromised left ventricular hemodynamics. It is recognized by persistent ST elevation in ECG and dyskinesia seen in echocardiography and radionuclide or contrast ventriculography. It may result in persistent left ventricular failure, arrhythmias and systemic embolism. Treatment consists of aneurysmectomy and associated coronary artery bypass surgery if so indicated.

b. Thromboembolism:

Formation of a thrombus within the left ventricle followed by systemic arterial embolism leading to occlusion of a peripheral artery requires immediate surgical embolectomy. Pulmonary embolism originates in the leg veins due to prolonged immobilization. These are prevented by anticoagulation. Thrombolytic therapy and embolectomy are occasionally required. The condition can be prevented by low molecular weight heparin.

KILLIPS CLASSIFICATION OF HEART FAILURE[98](Table 3)

Killip classified patients with MI into 4 classes based on findings of left ventricular failure by physical examination at time of admission into CCU.Hospital mortality from MI depends directly on severity of LV Dysfunction at time of admission.

Table 3

Class	Definition	Approx mortality(%)
Class I	Absence of rales over lung fields and absence of S3	8
Class II	Rales over 50% or less of lung fields or presence of S3	30
Class III	Rales over more than 50% of lung fields(pulmonary edema)	44
Class IV	Cardiogenic shock	80 to 100

Materials & Methods

MATERIALS AND METHODS

The present study is based on 100 cases of myocardial infarction admitted in I.C.C.U in Coimbatore medical college hospital during a period of one year.It is a descriptive study. Sampling was done by purposive non probability sampling .In each case , the following details were gathered and recorded .Age, sex ,occupation , diet , history of smoking , diabetes mellitus , hypertension and previous myocardial infarction were recorded . Thorough physical examination was done in each patient at the time of admission into the I.C.C.U. All the 100 cases were divided into 4 classes according to Killip's classification . Investigations like Hb, TC , DC, ESR, blood sugar, blood urea, serum creatinine, urine analysis, serum cholesterol , triglycerides and HDL were done and recorded in each case . ECG was done in all cases to confirm the diagnosis of myocardial infarction and to find out the type of infarction. In many cases, serial ECG's were taken to show the progress and prognosis. In many cases, Chest x-rays were taken.

Echocardiography evaluation was also done . Treatment particulars were carefully recorded and followed. Each patient was followed up during the stay in the hospital to assess the prognosis . Attempt has been made to compare the two types of infarctions – anterior and inferior wall

infarctions in relation to age, sex, occupation, risk factors and complications including with reference to Killip's classification .

Patients who had previous hospitalisation for ischemic heart disease were excluded from this study . Patients with non ST elevation myocardial infarction were excluded from the study. Patients who had both anterior and inferior wall myocardial infarction were also excluded from this study.

The relevant information was entered into a specially designed proforma. All the data collected through the proforma was analyzed through SPSS version 17 in terms of frequency, percentages and proportions.

Results and observation

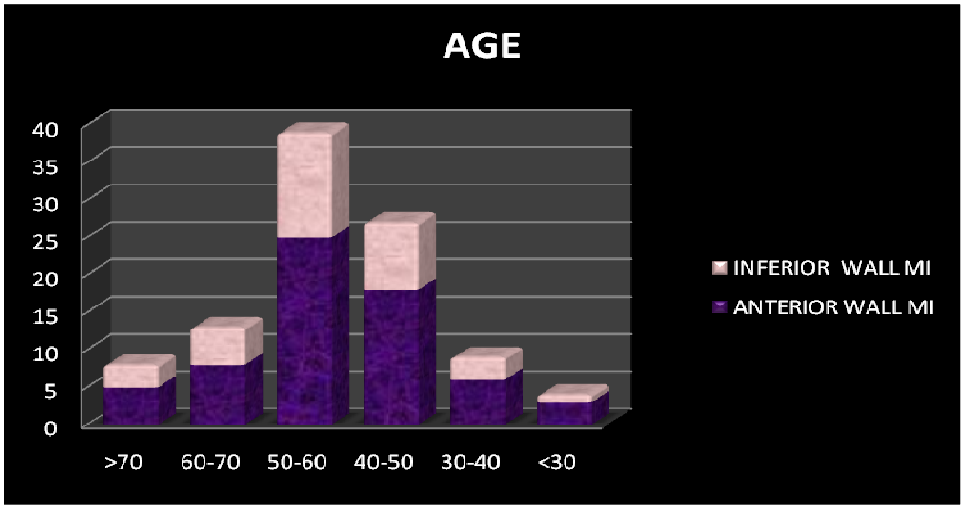
RESULTS AND OBSERVATION

INCIDENCE IN RELATION TO AGE (Graph 1)

AGE (yrs)	TOTAL	ANTERIOR WALL MI	INFERIOR WALL MI
>70	8	5(62.5%)	3(37.5%)
60-70	13	8(61.5%)	5(38.5%)
50-60	39	25(64.1%)	14(35.9%)
40-50	27	18(66.7%)	9(33.3%)
30-40	9	6(66.7%)	3(33.3%)
<30	4	3(75%)	1(25%)

Out of 100 patients,39 were in the 50- 60 age group , of which 25 (64.1%) patients were anterior wall MI and 14 (35.9%) were inferior wall MI.

Graph 1



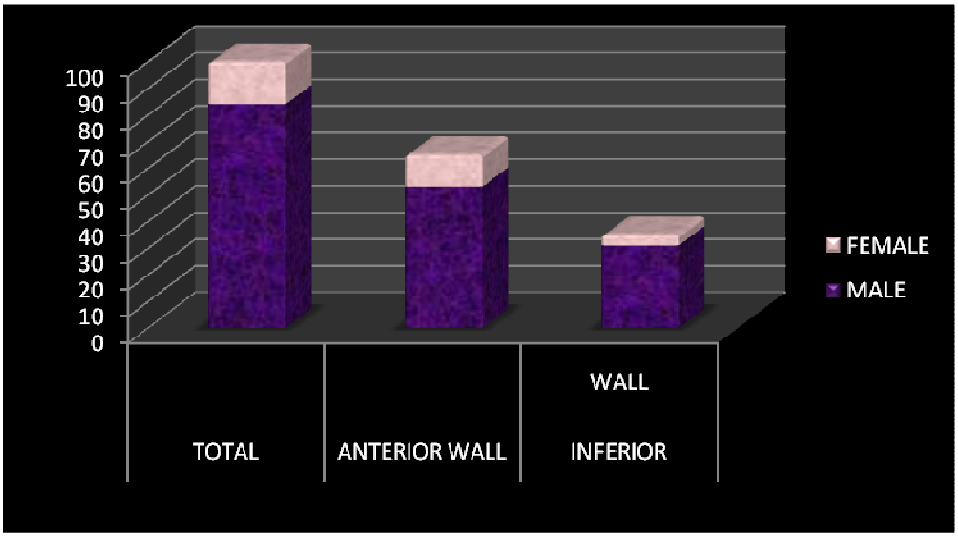
There is no significant difference between anterior and inferior wall MI in relation to age.

<u>INCIDENCE IN RELATION TO SEX</u>(Graph 2)

SEX	TOTAL	ANTERIOR WALL MI	INFERIOR WALL MI
MALE	84	53 (63.1%)	31 (36.9%)
FEMALE	16	12 (75%)	4 (25%)

Out of 100 patients,84 were male and 16 were female.Of the males,53 (63.1%) were anterior wall MI and 31(36.9%) were inferior wall MI. Of females, 12 (75%)were anterior wall MI and 4(25%) were inferior wall MI.

Graph 2



There is no significant difference between anterior wall MI and inferior wall MI in relation to sex.

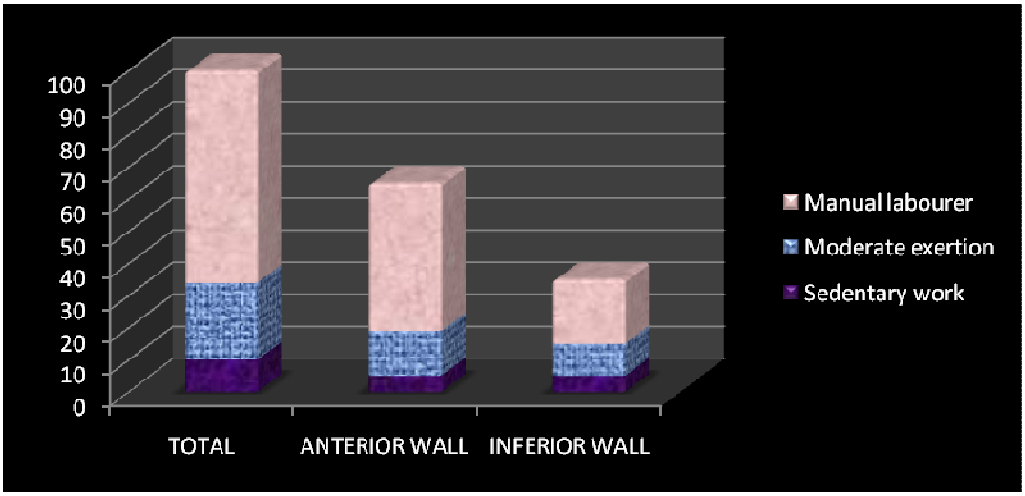
INCIDENCE IN RELATION TO OCCUPATION(Graph 3)

The patients were divided into 3 groups based on occupation

OCCUPATION	TOTAL	ANTERIOR WALL	INFERIOR WALL
Sedentary work	10	5(50%)	5(50%)
Moderate exertion	24	14(58.3%)	10(41.7%)
Manual labourer	66	46(69.7%)	20(30.3%)

Out of the 100 patients, 66 were manual labourers of which 46(69.7%) were anterior wall MI and 20 (30.3%)were inferior wall MI.

Graph 3



There is no significant difference between anterior and inferior wall MI in relation to occupation

INCIDENCE IN RELATION TO SITE OF INFARCTION(Graph 4,5)

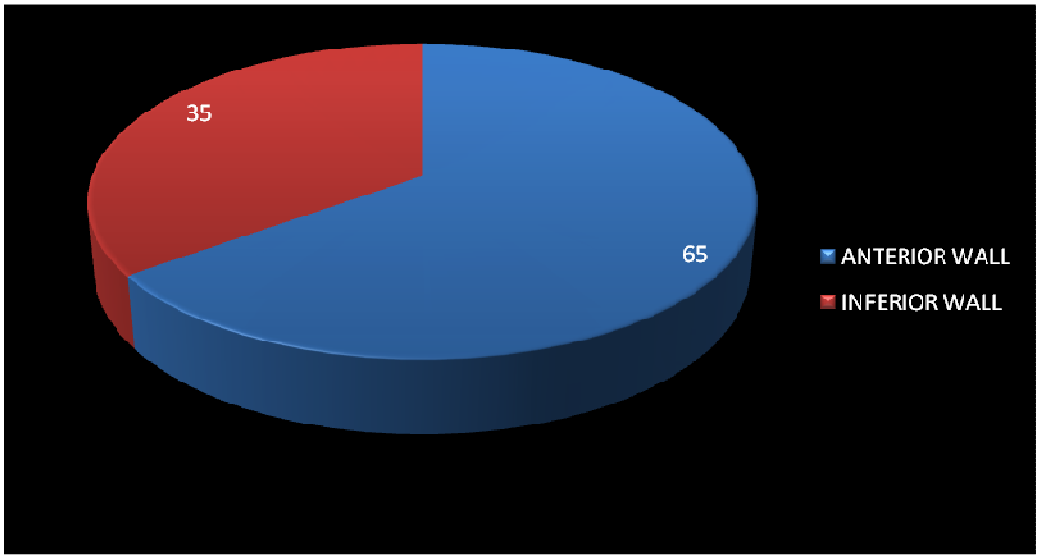
The site of infarction was determined by ECG analysis

ANATOMICAL ZONE	**ECG ZONE**
INFERIOR WALL	LII,III,Avf
ANTERO SEPTALWALL	V1-V4
EXTENSIVE ANTERIOR WALL	LI,AVL,V1-V6
ANTERO LATERAL WALL	LI,AVL,V5-V6

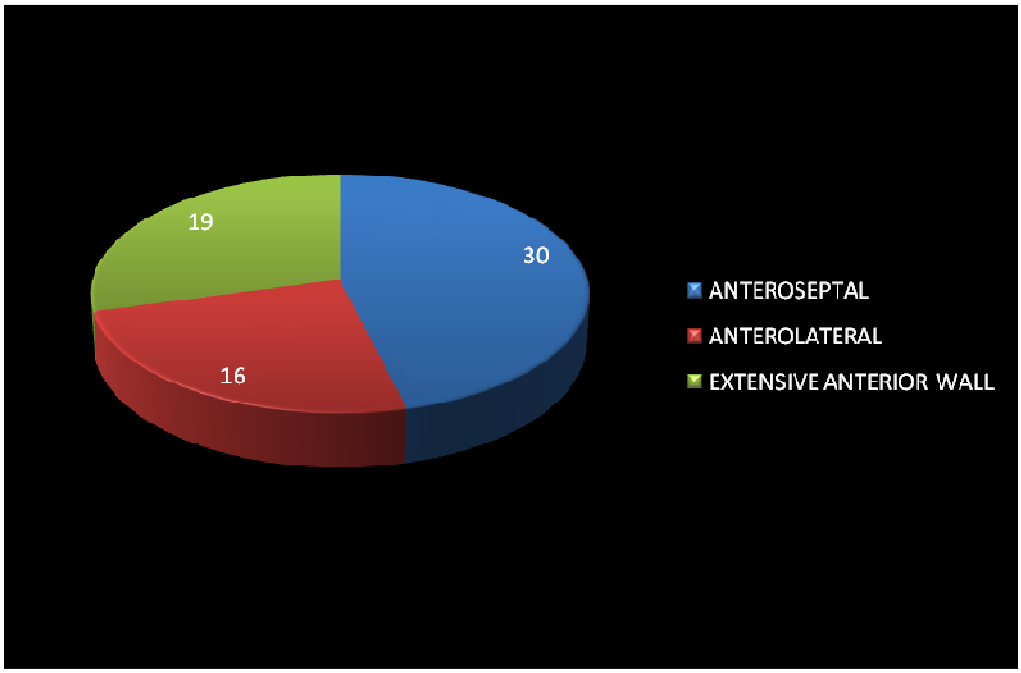
SITE OF INFARCTION	**NO: OF PATIENTS**
ANTERIOR WALL	65
ANTEROSEPTAL	30(46.2%)
ANTEROLATERAL	16(24.6%)
EXTENSIVE ANTERIOR WALL	19(29.2%)
INFERIOR WALL	35

Out of the 100 patients, 65 were anterior wall MI and 35 were inferior wall MI.Out of the anterior wall MI patients, 30 (46.2%)were anteroseptal,16(24.6%) were anterolateral and 19(29.2%) were extensive anterior wall MI.

Graph 4



Graph 5



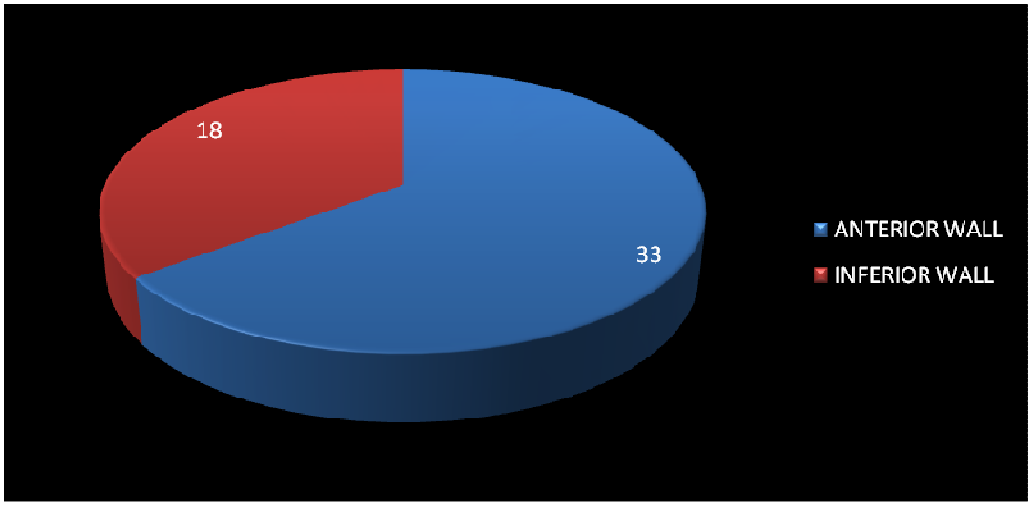
HYPERTENSION(Graph 6)

The role of hypertension as a risk factor was assessed .

HYPERTENSION	TOTAL	ANTERIOR WALL	INFERIOR WALL
TOTAL	51	33(65%)	18(35%)
MALE	41	27(66%)	14(34%)
FEMALE	10	6(60%)	4(40%)

Out of the 51 patients with hypertension , 33 were anterior wall MI and 18 were inferior wall MI.

Graph 6



There is no significant difference between anterior wall and inferior wall MI in relation to hypertension as a risk factor

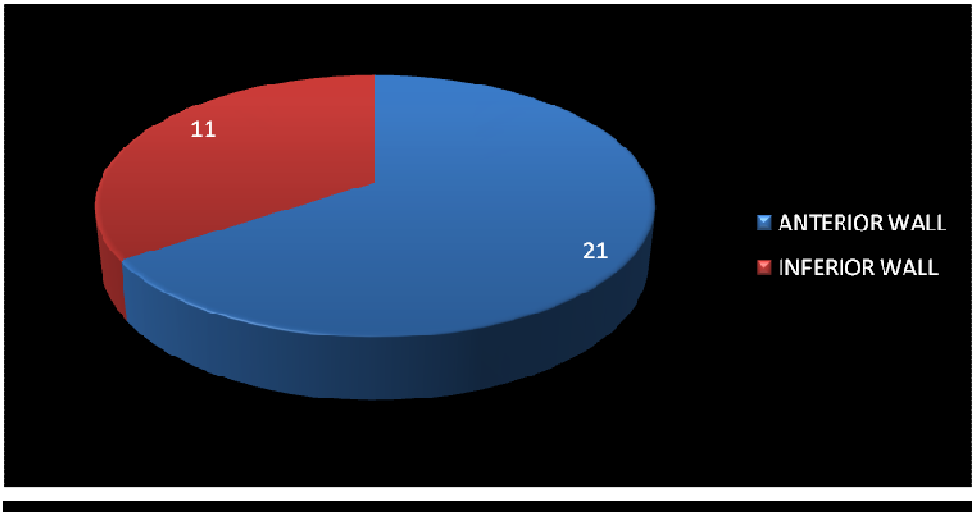
DIABETES(Graph 7)

The role of diabetes as a risk factor was also studied.

DIABETES	TOTAL	ANTERIOR WALL	INFERIOR WALL
TOTAL	32	21(66%)	11(34%)
MALE	24	16(66.7%)	8(33.3%)
FEMALE	8	5(62.5%)	3(37.5%)

Out of the 32 patients with diabetes ,21 were anterior wall MI and 11 were inferior wall M1.

Graph 7



There is no significant difference between anterior wall and inferior wall MI in relation to diabetes as a risk factor.

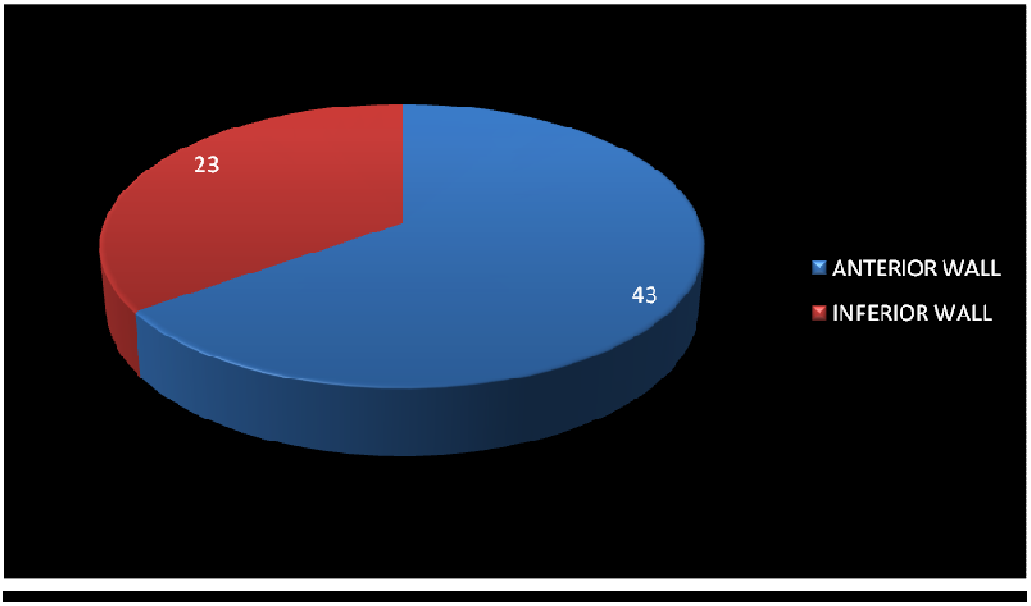
SMOKING(Graph 8)

The role of smoking as a risk factor was also assessed

	TOTAL	ANTERIOR WALL	INFERIOR WALL
SMOKER	66	43(65%)	23(35%)

Out of 84 males , 66(78.6%) were smokers .Out of 66 male smokers, 43 were anterior wall MI and 23 were inferior wall MI.

Graph 8



There is no significant difference between anterior wall and inferior wall MI in relation to smoking as a risk factor.

BODY MASS INDEX (BMI)(Graph 9)

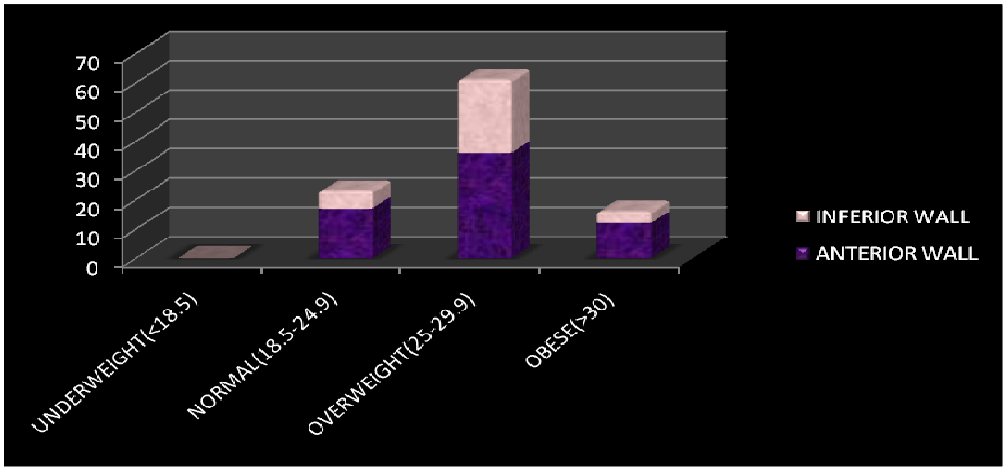
The Body Mass Index (BMI) was calculated by dividing the patient's body weight in kg by the square of patient's height in metres.

BMI = Weight (kg)/[Height(m)]^2. According to patient's BMI,they were divided into 4 groups

BMI	TOTAL	ANTERIOR WALL	INFERIOR WALL
UNDERWEIGHT(<18.5)	0	0	0
NORMAL(18.5-24.9)	23	17(74%)	6(26%)
OVERWEIGHT(25-29.9)	61	36(59%)	25(41%)
OBESE(>30)	16	12(75%)	4(25%)

Out of the 100 patients ,61 were overweight ,of which 36 were anterior wall MI and 25 were inferior wall MI.16 patients were obese ,of which 12 were anterior wall MI and 4 were inferior wall MI.

Graph 9



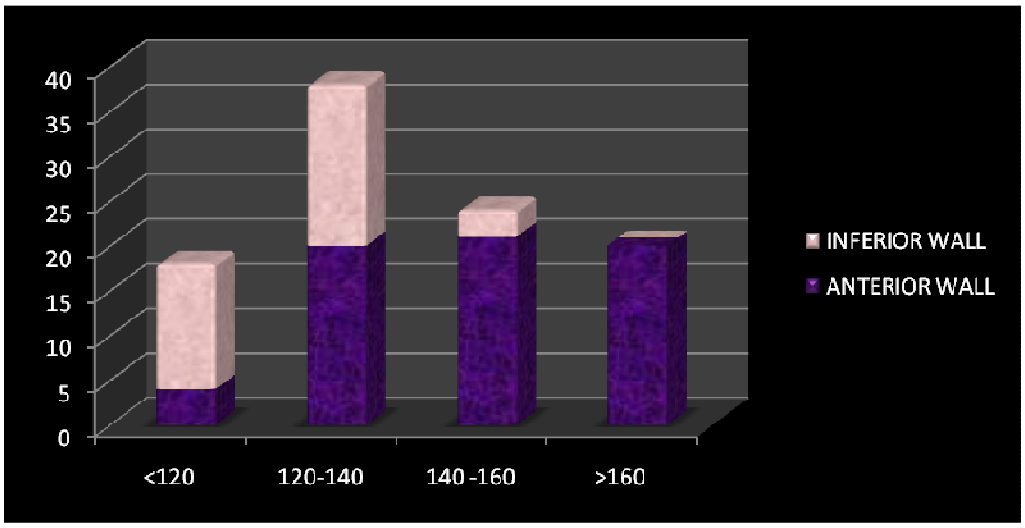
There is no significant difference between anterior wall and inferior wall MI in relation to BMI as a risk factor.

SYSTOLIC BP (Graph 10)

SBP (mmHg)	TOTAL	ANTERIOR WALL	INFERIOR WALL
<120	18	4(22.2%)	14(77.8%)
120-140	38	20(52.6%)	18(47.4%)
140 -160	24	21(87.5%)	3(12.5%)
>160	20	20(100%)	0

Out of the 100 patients, 38 had SBP between 120 and 140, of which 20 were anterior wall MI and 18 were inferior wall MI.

Graph 10



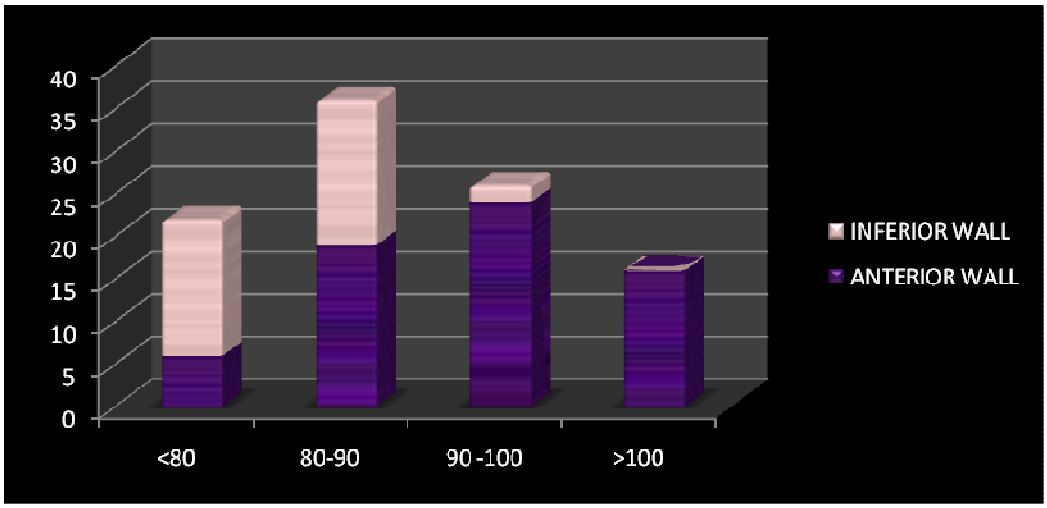
There is a significant difference in systolic BP between anterior and inferior wall MI with a chi square value of 33.14 and p value < 0.001.

DIASTOLIC BP(Graph 11)

DBP (mmHg)	TOTAL	ANTERIOR WALL	INFERIOR WALL
<80	22	6(27.3%)	16(72.7%)
80-90	36	19(52.8%)	17(47.2%)
90 -100	26	24(92.3%)	2(7.7%)
>100	16	16(100%)	0

Out of the 100 patients,36 had DBP between 80 and 90, of which 19 were anterior wall MI and 17 were inferior wall MI.

Graph 11



There is a significant difference in diastolic BP between anterior and inferior wall MI with a chi square value of 30.91 and p value <0.001.

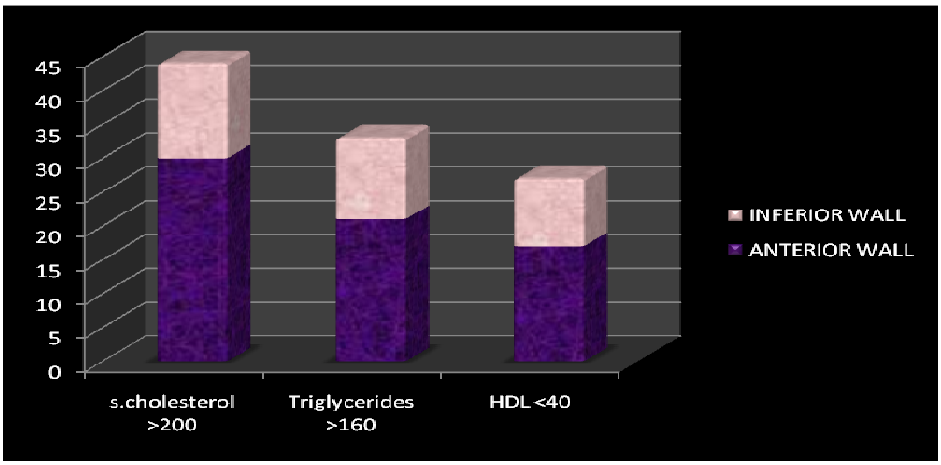
HYPERLIPIDEMIA(Graph 12)

Hyperlipidemia as a risk factor was studied by measuring the lipid profile . Total cholesterol >200mg/dl,triglycerides.160 mg/dl,HDL < 40 mg/dl were considered as risk factors.

LIPID PROFILE (mg/dl)	TOTAL	ANTERIOR WALL	INFERIOR WALL
s.cholesterol >200	44	30(68.2%)	14(31.8%)
Triglycerides >160	33	21(63.6%)	12(36.4%)
HDL <40	27	17(63%)	10(37%)

44 patients had hypercholesterolemia, of which 30 were anterior wall and 14 were inferior wall MI. 33 patients had hypertriglyceridemia of which 21 were anterior wall and 12 were inferior wall MI. 27 patients had low HDL of which 17 were anterior wall and 10 were inferior wall MI.

Graph 12



There is no significant difference between anterior wall and inferior wall MI in relation to lipid profile as a risk factor.

RISK FACTORS

RISK FACTORS	SITE	N	MEAN	Std deviation	t	p
BMI	AW	65	26.99	2.665	0.341	0.734
	IW	35	27.16	2.056		
SBP	AW	65	147.75	20.909	7.298	<0.001
	IW	35	115.20	21.953		
DBP	AW	65	93.26	10.946	7.321	<0.001
	IW	35	76.63	10.625		
Total cholesterol	AW	65	203.95	35.485	0.441	0.660
	IW	35	200.77	32.249		
Triglycerides	AW	65	149.78	18.729	0.783	0.436
	IW	35	152.80	17.691		
HDL	AW	65	44.97	8.595	0.400	0.690
	IW	35	44.23	9.277		

The t value 7.298 for the mean difference in systolic BP between anterior and inferior wall MI is significant ($p<0.001$).It reveals that patients with anterior wall MI have higher systolic BP when compared to inferior wall MI.

The t value 7.321 for the mean difference in diastolic BP between anterior and inferior wall MI is significant ($p<0.001$).It reveals that patients with anterior wall MI have higher diastolic BP when compared to inferior wall MI.

The effect of the other risk factors was not significant.

KILLIP'S CLASSIFICATION(Graph 13)

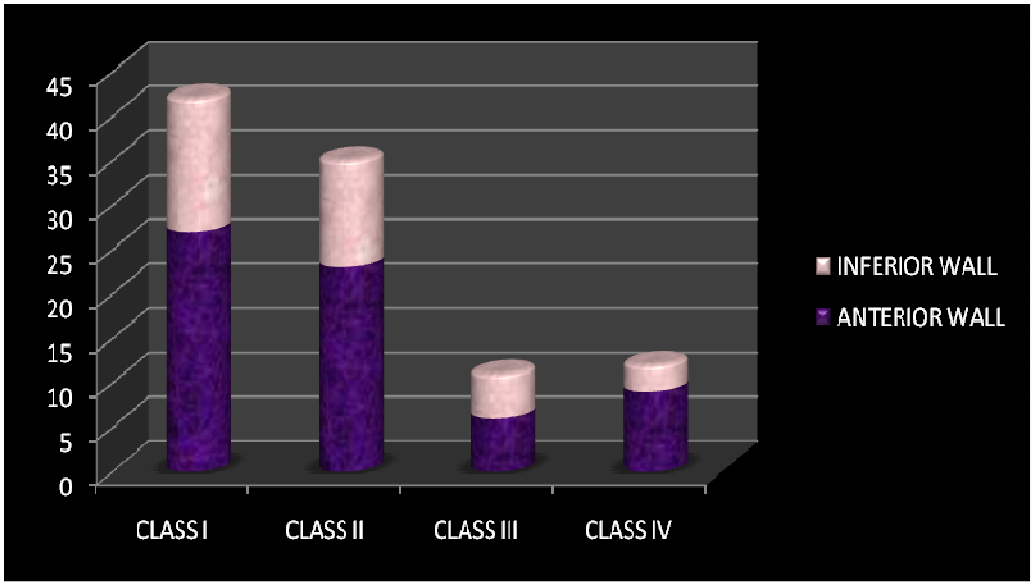
The patients were divided into 4 classes based on Killip's classification

Class	Definition	Approx mortality(%)
Class I	Absence of rales over lung fields and absence of S3	8
Class II	Rales over 50% or less of lung fields or presence of S3	30
Class III	Rales over more than 50% of lung fields(pulmonary edema)	44
Class IV	Cardiogenic shock	80 to 100

KILLIP'S CLASS	TOTAL	ANTERIOR WALL	INFERIOR WALL
CLASS I	42	27(65.3%)	15(34.7%)
CLASS II	35	23(65.7%)	12(34.3%)
CLASS III	11	6(55.5%)	5(45.5%)
CLASS IV	12	9(75%)	3(25%)

Out of the 100 patients, 42 were in class I, of which 27 were anterior wall MI and 15 were inferior wall MI.35 were in class II ,of which 23 were anterior wall and 12 were inferior wall MI. 11 were in class III, of which 6 were anterior wall and 5 were inferior wall MI.12 were in class IV , of which 9 were anterior wall and 3 were inferior wall MI.

Graph 13



There is no significant difference between anterior wall and inferior wall MI in relation to Killip's classification.

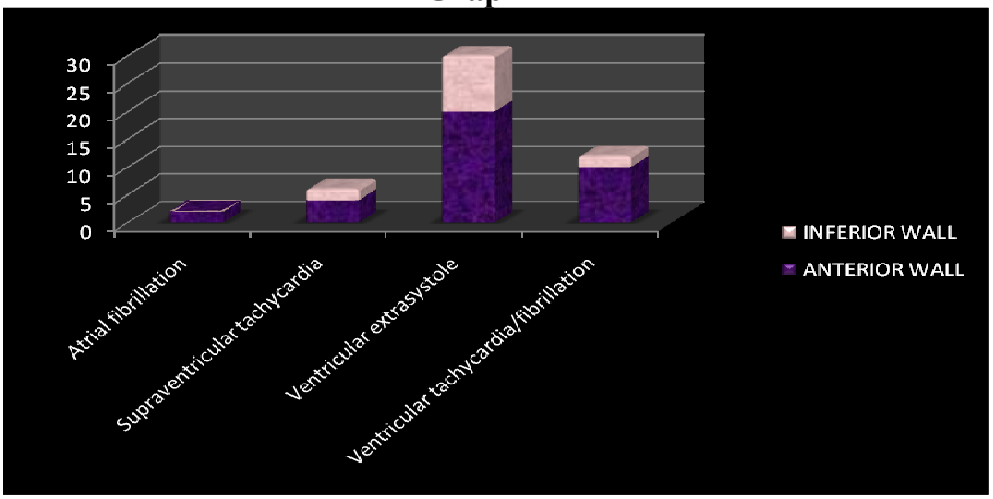
TACHYARRHYTHMIAS(Graph 14)

The incidence of various types of tachyarrhythmias was assessed.

TACHYARRHYTHMIA	TOTAL	ANTERIOR WALL	INFERIOR WALL
Atrial fibrillation	2(4%)	2(100%)	0
Supraventricular tachycardia	6(12%)	4(66.7%)	2(33.3%)
Ventricular extrasystole	30(60%)	20(66.7%)	10(33.3%)
Ventricular tachycardia/fibrillation	12(24%)	10(83.3%)	2(16.7%)
TOTAL	**50**	**36(72%)**	**14(28%)**

Out of the 50 patients with tachyarrhythmias, 36 were anterior wall MI and 14 were inferior wall MI.30 had ventricular extrasystole, of which 20 were anterior wall MI and 10 were inferior wall MI.

Graph 14



Tachyarrhythmias were more common in anterior wall MI than in inferior wall MI .

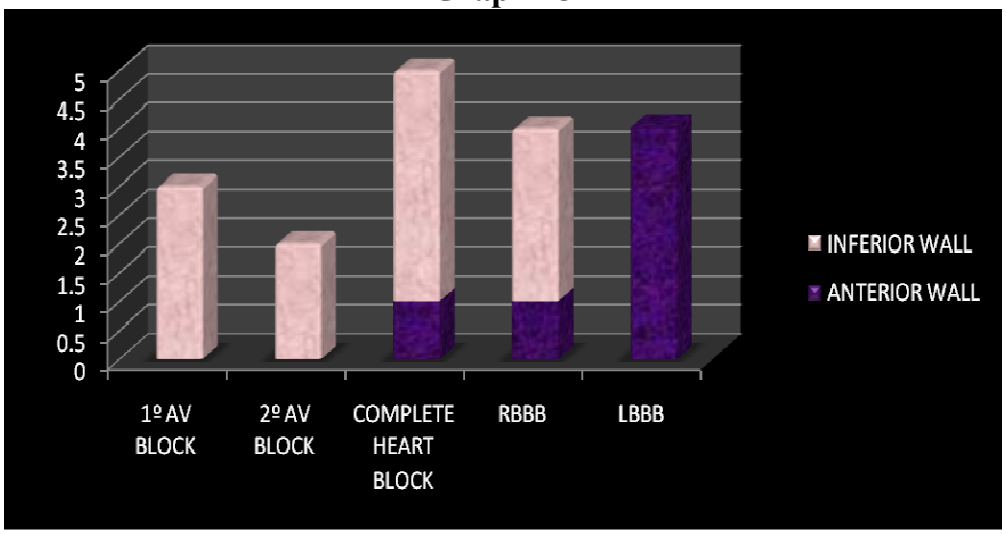
HEART BLOCK(Graph 15)

The incidence of various heart blocks was assessed.

HEART BLOCK	TOTAL	ANTERIOR WALL	INFERIOR WALL
1º AV BLOCK	3(16.7%)	0	3(100%)
2º AV BLOCK	2(11.1%)	0	2(100%)
COMPLETE HEART BLOCK	5(27.8%)	1(20%)	4(80%)
RBBB	4(22.2%)	1(25%)	3(75%)
LBBB	4(22.2%)	4(100%)	0
TOTAL	18	6(33.3%)	12(66.7%)

Out of the 18 patients with heart blocks,12 were inferior wall MI and 6 were anterior wall MI.

Graph 15



Heart blocks were more common in inferior wall MI than in anterior wall MI.

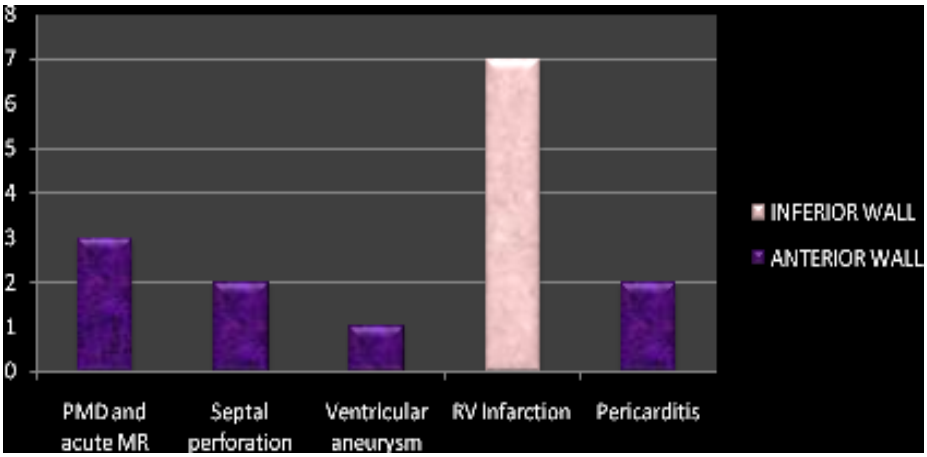
<u>OTHER COMPLICATIONS</u>(Graph 16)

Other complications were also assessed.

COMPLICATIONS	TOTAL	ANTERIOR WALL	INFERIOR WALL
PMD and acute MR	3(20%)	3(100%)	0
Septal perforation	2(13.3%)	2(100%)	0
Ventricular aneurysm	1(6.7%)	1(100%)	0
RV Infarction	7(46.7%)	0	7(100%)
Pericarditis	2(13.3%)	2(100%)	0
TOTAL	15	8(53.3%)	7(46.7%)

Out of the 15 other complications, 8 were anterior wall MI and 7 were inferior wall MI.

Graph 16



Anterior wall MI had more incidence of other complications when compared to inferior wall MI .

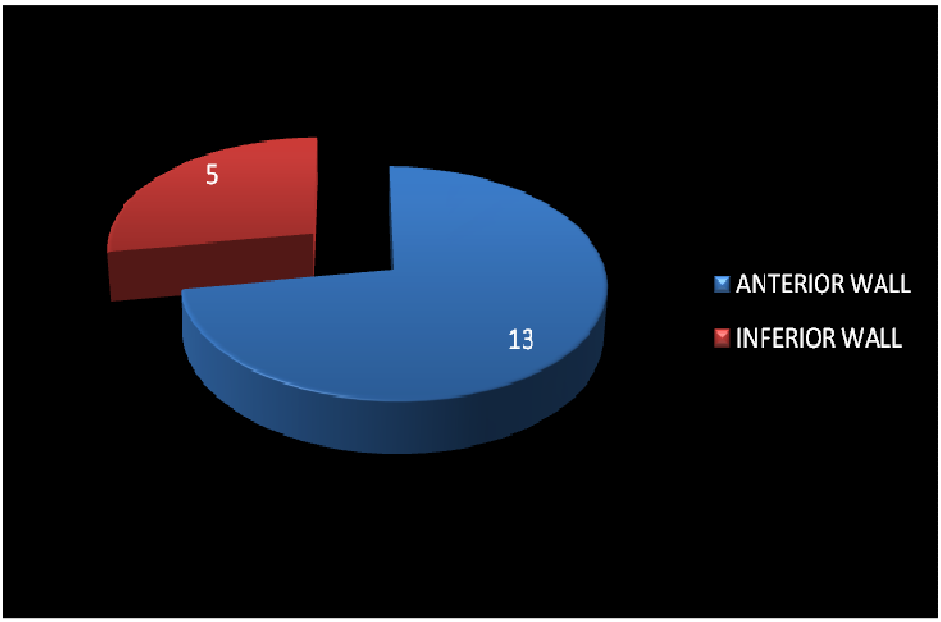
MORTALITY

The mortality of the patients was studied in relation to age ,sex,arrhythmias and killip class.(**Graph 17**)

TOTAL DEATHS	ANTERIOR WALL	INFERIOR WALL
18	13(72.2%)	5(27.8%)

Out of the 18 deaths,13 were anterior wall MI and 5 were inferior wall MI.

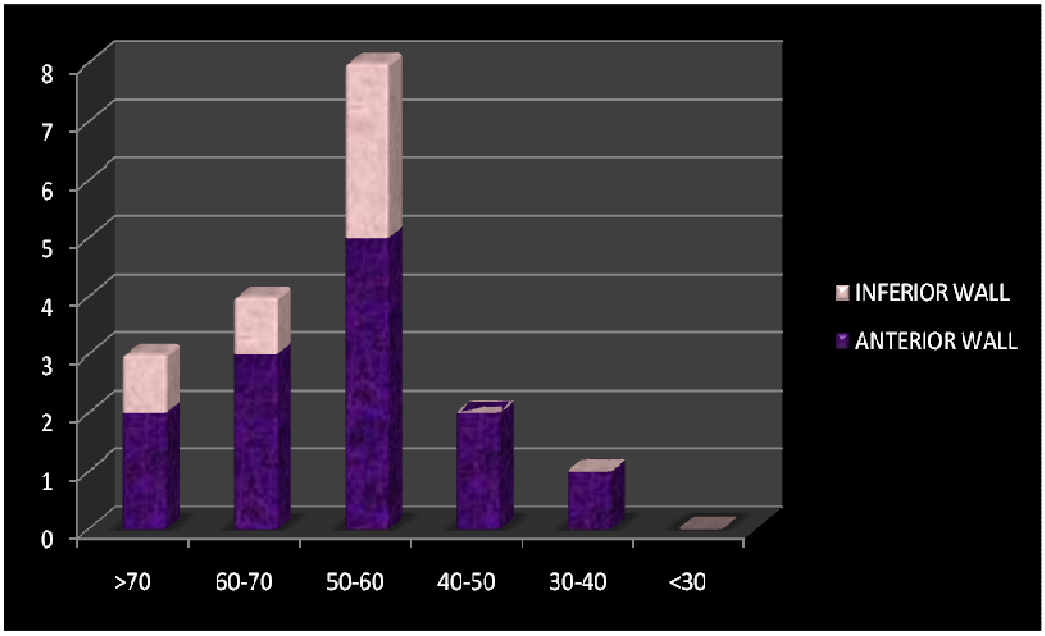
Graph 17



AGE (yrs)	TOTAL	ANTERIOR WALL	INFERIOR WALL
>70	3(16.7%)	2(66.7%)	1(33.3%)
60-70	4(22.2%)	3(75%)	1(25%)
50-60	8(44.4%)	5(62.5%)	3((37.5%)
40-50	2(11.1%)	2(100%)	0
30-40	1(5.6%)	1(100%)	0
<30	0	0	0

Out of the 18 deaths,8 were between 50 and 60 , of which 5 were anterior wall MI and 3 were inferior wall MI.

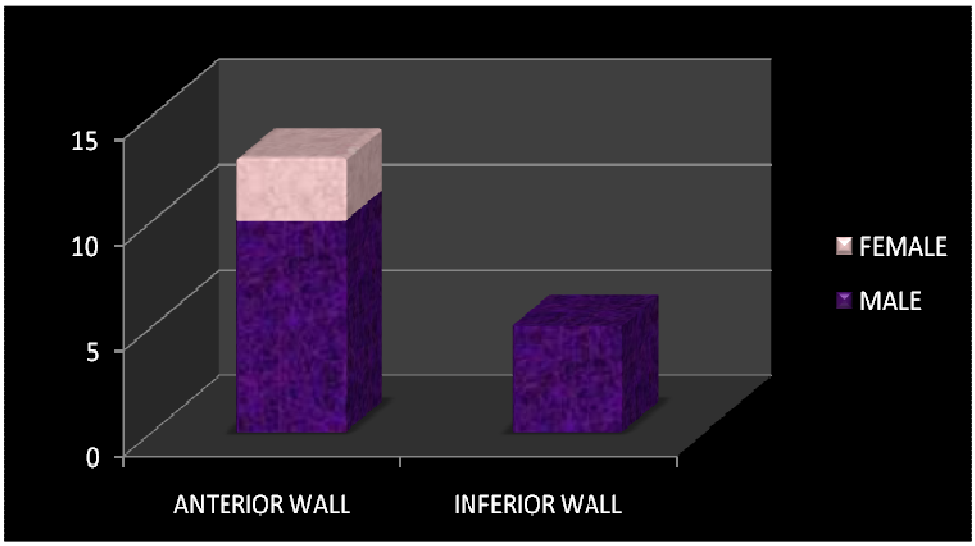
Graph 18



SEX	TOTAL	ANTERIOR WALL	INFERIOR WALL
MALE	15(83.3%)	10(66.7%)	5(33.3%)
FEMALE	3(16.7%)	3(100%)	0

Out of the 18 deaths, 15 were male,of which 10 were anterior wall and 5 were inferior wall MI.

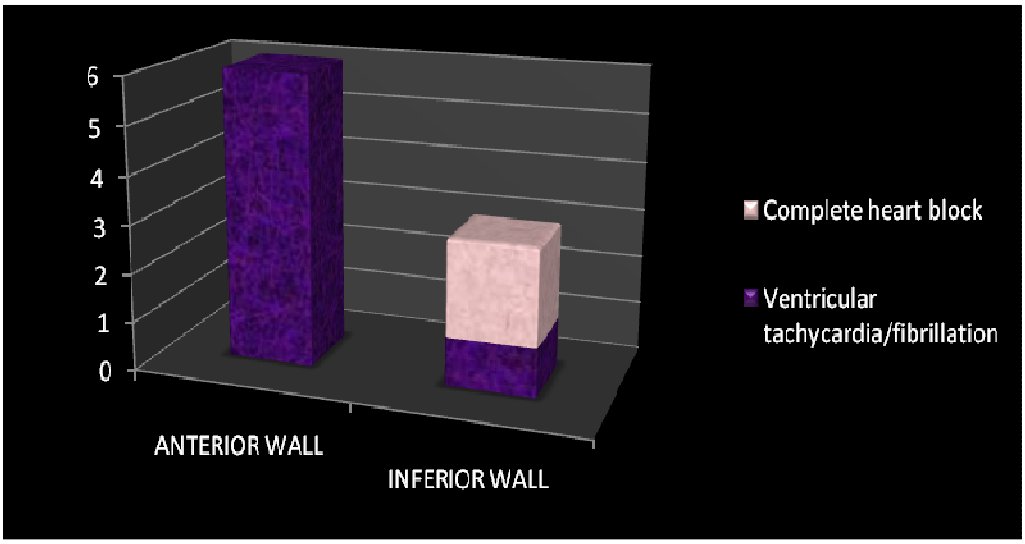
Graph 19



ARRHYTHMIA	TOTAL	ANTERIOR WALL	INFERIOR WALL
Ventricular tachycardia/fibrillation	7(88.9%)	6(85.7%)	1(14.3%)
Complete heart block	2(11.1%)	0	2(100%)

Out of the 18 deaths, 7 were due to ventricular tachycardia/ventricular fibrillation, of which 6 were anterior wall and 1 was inferior wall MI.

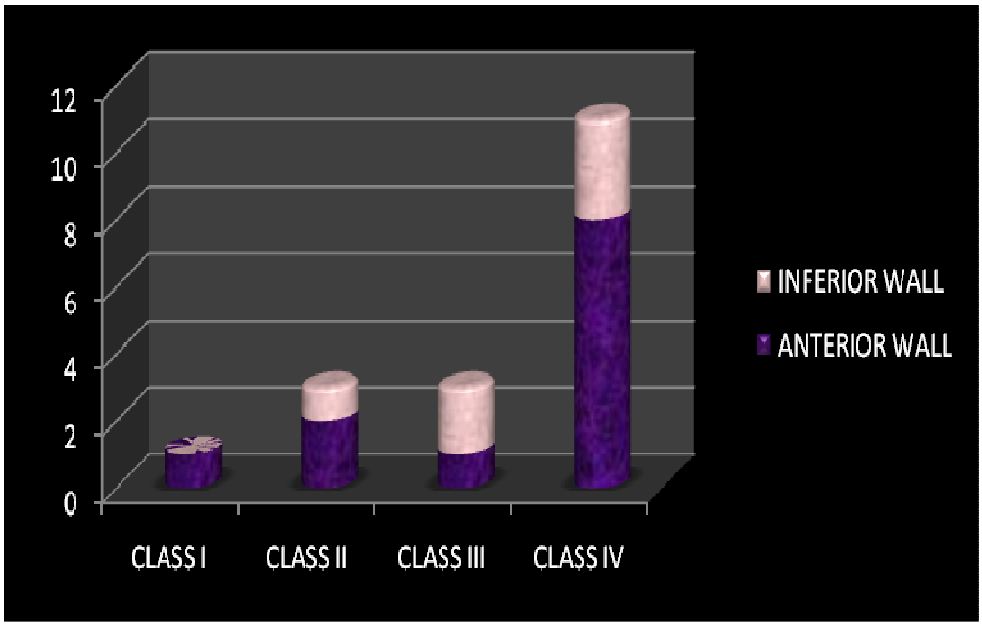
Graph 20



KILLIP CLASS	TOTAL	ANTERIOR WALL	INFERIOR WALL
CLASS I	1(5.5%)	1(100%)	0
CLASS II	3(16.7%)	2(66.7%)	1(33.3%)
CLASS III	3(16.7%)	1(33.3%)	2(66.7%)
CLASS IV	11(61.1%)	8(72.7%)	3(27.3%)

11 deaths were in killip class IV ,of which 8 were anterior wall and 3 were inferior wall MI.

Graph 21



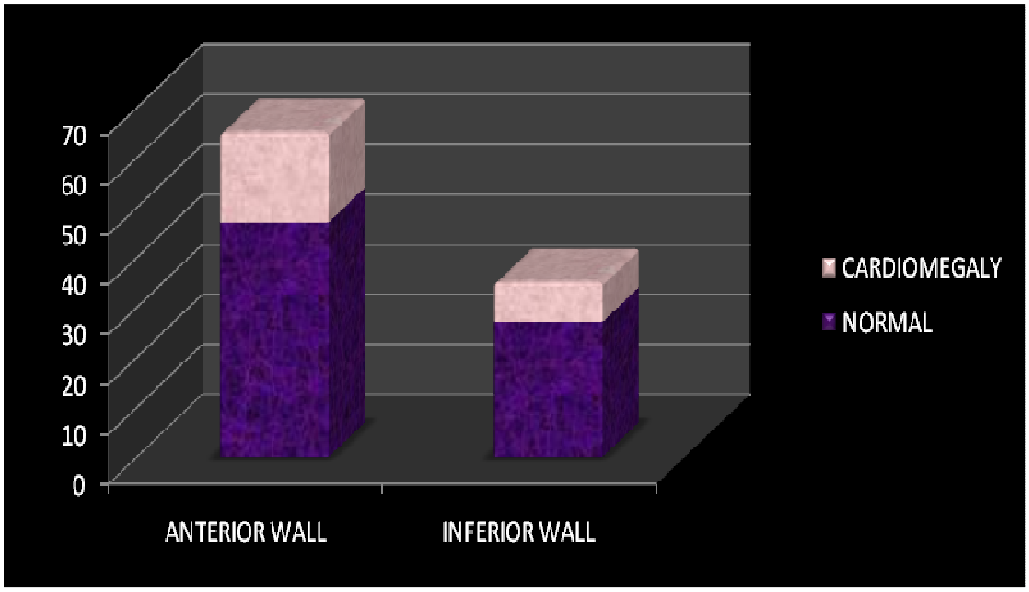
CHEST X RAY

Chest X ray was reported as normal or cardiomegaly.

CXR	TOTAL	ANTERIOR WALL	INFERIOR WALL
NORMAL	74	47(63.5%)	27(36.5%)
CARDIOMEGALY	26	18(69.2%)	8(30.8%)

26 patients had cardiomegaly ,of which 18 were anterior wall and 8 were inferior wall MI.

Graph 22



More of anterior wall MI patients had cardiomegaly when compared to inferior wall MI.

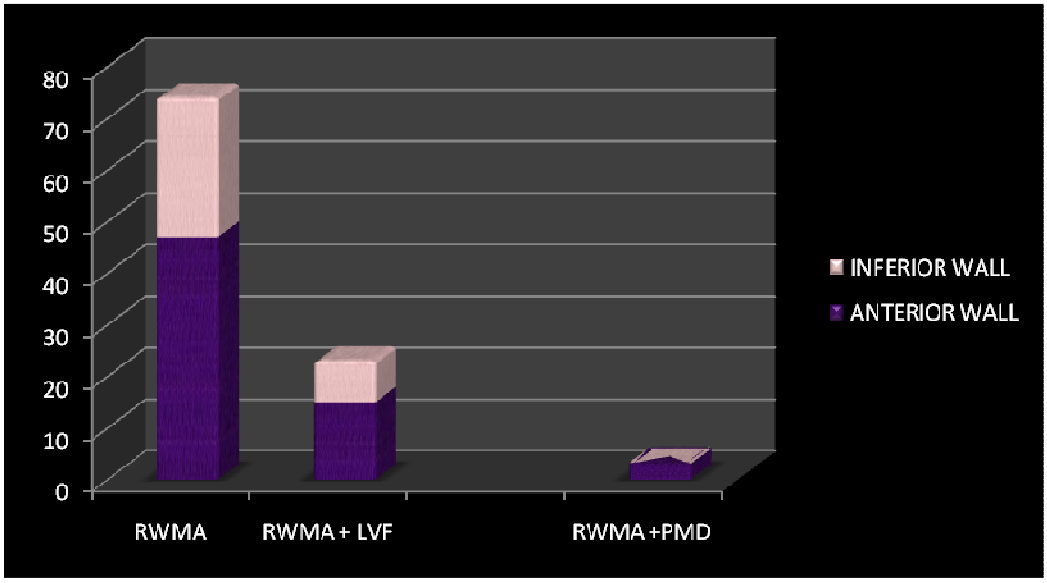
ECHOCARDIOGRAM (Graph 23)

Echo was taken for the patients and grouped according to echo findings

ECHO FINDING	TOTAL	ANTERIOR WALL	INFERIOR WALL
RWMA	74	47(63.5%)	27(36.5%)
RWMA + LVF	23	15(65.2%)	8(34.8%)
RWMA +PMD	3	3(100%)	0

RWMA was present in 74 patients of which 47 were anterior wall and 27 were inferior wall MI

Graph 23



More of anterior wall MI patients had RWMA when compared to inferior wall MI.

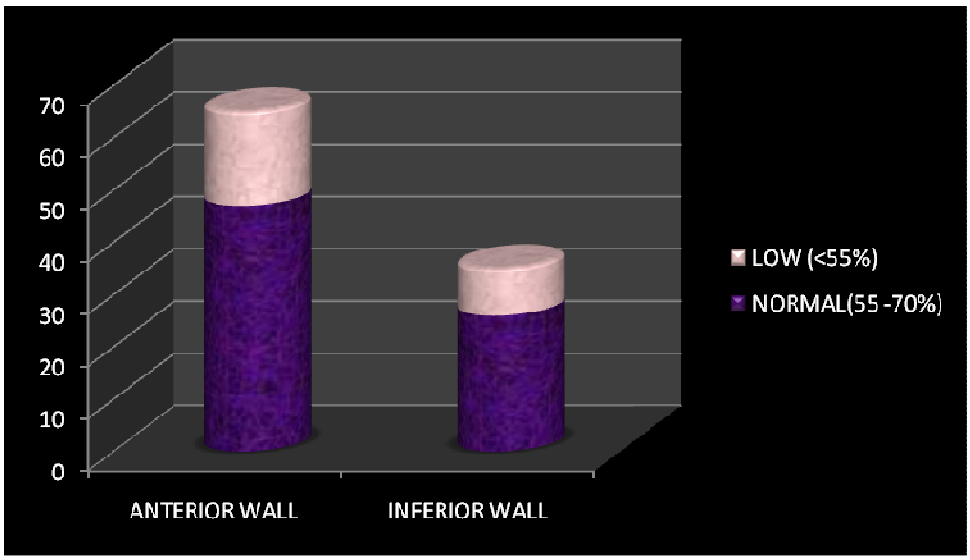
EJECTION FRACTION(EF)(Graph 24)

Ejection fraction was estimated using echo.

EJECTION FRACTION (%)	TOTAL	ANTERIOR WALL	INFERIOR WALL
NORMAL(55 - 70%)	73	47(64.4%)	26(35.6%)
LOW (<55%)	27	18(66.7%)	99(33.3%)

27 patients had low EF, of which 18 were anterior wall and 9 were inferior wall MI.

Graph 24



More of anterior wall MI patients had low EF when compared to inferior wall MI.

Discussion

DISCUSSION

Anterior wall MI is generally considered to have a poor prognosis and higher mortality when compared to inferior wall MI. In the present study, anterior and inferior wall MI patients are compared in terms of age, sex ,risk factors, complications and mortality.

In relation to age, Age is a definite unmodifiable risk factor. Atherosclerosis develops progressively as age advances. According to Prasanth etal[53] , South Asians develop AMI at an earlier age when compared to other countries and the mean age was 53 years. In my study, AMI was highest in the age group of 50-60 yrs(39%) ,which was comparable with the above mentioned study. But there was no significant difference between anterior wall and inferior wall MI in relation to age.

In relation to sex, Men are more affected than premenopausal women. However after menopause the incidence of atheroma rises in women. According to Sonia etal[55] , MI occurs more in males and earlier in males. In my study also, the incidence of MI was more in males (84%) which was comparable with the above mentioned study. But there was no significant difference between anterior wall and inferior wall MI in relation to sex .

In relation to occupation, previously sedentary work used to be considered a risk factor for MI, but now even heavy workers are being affected. In my study, manual labourers (66%) have highest incidence of MI , which is correlating. But there was no significant difference between anterior wall and inferior wall MI in relation to occupation .

In relation to site, according to Peter etal[99] , anterior wall MI is more common than inferior wall MI. In my study also, anterior wall MI (65%) was more common than inferior wall MI. Out of the anterior wall MI patients, majority were anteroseptal (46.2%) .

In relation to hypertension , the incidence of coronary heart disease increases as blood pressure rises and the excess risk is related to both systolic and diastolic blood pressure . According to Trenkwalder etal[67], Hypertension is a significant risk factor for MI and associated morbidity and mortality.In my study also ,hypertension was an important risk factor and was present in 51% of the patients. Out of this, majority(65%) were anterior wall MI. But there was no significant difference between anterior wall and inferior wall MI in relation to hypertension as a risk factor.

In relation to diabetes, Diabetes Mellitus is a coronary heart disease risk equivalent . According to Lundberg etal[74], Diabetes is a significant risk factor for MI and associated morbidity and mortality. In my study

,Diabetes was present in 32% of cases, out of which, majority were anterior wall MI (66%). So diabetes was a significant risk factor for MI. But there was no significant difference between anterior wall and inferior wall MI in relation to diabetes as a risk factor.

In relation to smoking, there is a strong, consistent and dose linked relationship between cigarette smoking and Ischaemic heart disease . According to Prescott etal[62] , smoking is a significant risk factor for MI and associated morbidity and mortality. In my study , 66(78.6%) of the males were smokers, out of which 43(65%) were anterior wall MI .So smoking was a significant risk factor for MI. But there was no significant difference between anterior wall and inferior wall MI in relation to smoking as a risk factor.

In relation to obesity , particularly the male pattern of centripetal or visceral fat accumulation is probably an independent risk factor for CHD. According to Lavie etal[80] ,Obesity is a significant risk factor for MI and associated morbidity and mortality . In my study, BMI was used for assessing obesity and 61% of the patients were overweight ,of which 66% were anterior wall MI.16% of the patients were obese ,of which 75% were anterior wall MI .So obesity was a significant risk factor for MI . But there was no significant difference between anterior wall and inferior wall MI in relation to BMI as a risk factor.

In relation to Systolic BP ,anterior wall MI patients have higher SBP where as inferior wall MI patients have lower SBP usually because of vagal stimulation. In my study , 38 % of the patients had SBP between 120 and 140, of which 20 (52.6%) were anterior wall MI. The mean SBP of AWMI patients was 147.75 mmHg and the mean SBP of IWMI patients was 93.26 mmHg. So there was a significant difference in systolic BP between anterior and inferior wall MI with a chi square value of 33.14 and p value < 0.001, which was comparable with previous studies.

In relation to Diastolic BP, anterior wall MI patients have higher DBP where as inferior wall MI patients have lower DBP usually because of vagal stimulation . In my study, 36% of the patients had DBP between 80 and 90, of which 19(52.8%) were anterior wall MI . The mean DBP of AWMI patients was 115.20 mmHg and the mean DBP of IWMI patients was 76.63 mmHg. So there was a significant difference in diastolic BP between anterior and inferior wall MI with a chi square value of 30.91 and p value <0.001, which was comparable with previous studies.

In relation to lipid profile, A wealth of evidence from epidemiological, clinical and experimental studies has established the association between hyperlipidemia and atherosclerosis.According to Goldstein etal[71] , hypercholesterolemia and hypertriglyceridemia are

important risk factors for MI . In my study , 44% of the patients had hypercholesterolemia, of which 30 (68.2%) were anterior wall MI, 33% of the patients had hypertriglyceridemia of which 21(63.6%) were anterior wall MI and 27% of the patients had low HDL of which 17(63%) were anterior wall MI. So hypercholesterolemia, hypertriglyceridemia and low HDL were significant risk factors for MI. But there was no significant difference between anterior wall and inferior wall MI in relation to lipid profile as a risk factor.

In relation to the various risk factors, the t value 7.298 for the mean difference in systolic BP between anterior and inferior wall MI was significant ($p<0.001$). It revealed that patients with anterior wall MI have higher systolic BP when compared to inferior wall MI .The t value 7.321 for the mean difference in diastolic BP between anterior and inferior wall MI was also significant ($p<0.001$). It revealed that patients with anterior wall MI have higher diastolic BP when compared to inferior wall MI. The effect of the other risk factors was not significant.

In relation to Killip's Classification of heart failure, According to the original Killip study[98] , majority of the patients were in class II (38%).In my study, majority of the patients were in class I (42%), of which 27(65.3%) were anterior wall MI .This difference might be because of better awareness of patients and early diagnosis and treatment

. But there was no significant difference between anterior wall and inferior wall MI in relation to Killip's classification.

In relation to arrhythmias, in general tachyarrhythmias are common in anterior wall MI, whereas bradyarrhythmias are more common in inferior wall MI.According to Siddique etal[93], most common tachyarrhythmia in MI patients is ventricular extrasystole (56%). In my study, 50% of the patients had tachyarrhythmias, of which 36 (72%) were anterior wall MI. 30 (60%) had ventricular extrasystole, of which 20(66.7%) were anterior wall MI. So tachyarrythmias were more common in anterior wall MI and most common tachyarrythmia was ventricular extrasystole. This was comparable with the above mentioned study.

In relation to heart blocks,in general heart blocks are more common in inferior wall MI. According to Siddique etal[93], most common heart block in MI patients is complete heart block. In my study,18% of patients had heart blocks, of which 12 (66.7%)were inferior wall MI. So heart blocks were more common in inferior wall MI and most common heart block was complete heart block (27.8%) .This was comparable with the above mentioned study.

In relation to the other complications, in general anterior wall MI has more complications when compared to inferior wall MI. Stone etal[97]

said that patients with anterior infarction had a substantially worse in-hospital and follow-up clinical course compared with those with inferior infarction, evidenced by a larger infarct size , lower admission left ventricular ejection fraction , higher incidence of heart failure , serious ventricular ectopic activity , in-hospital death and total cumulative cardiac mortality.In my study, 15 % of patients had other complications,of which 8 (53.3%)were anterior wall MI. The most common other complication was RV infarction. So anterior wall MI had more incidence of other complications when compared to inferior wall MI.

In relation to mortality, in general, anterior wall MI has a higher mortality when compared to inferior wall MI.Stone etal[97] said that anterior wall MI has more mortality than inferior wall MI. In my study,18% of the patients died , of which 13(72.2%) were anterior wall MI .Out of the deaths, 8 (44.4%)were between 50 and 60 years of age , of which 5(62.5%) were anterior wall MI .15(83.3%) deaths were males of which 10(66.7%) were anterior wall MI. 7 (88.9%)deaths were due to VT/VF,of which 6 (85.7%) were anterior wall MI.11 (61.1%)deaths were in killip class IV ,of which 8 were anterior wall MI. In killip original study , the mortality was 67% for killip class IV, which was comparable.

So anterior wall MI had more mortality when compared to inferior wall MI. The above observations were comparable with previous studies.

In relation to Chest X Ray,28 patients had cardiomegaly ,of which 18(69.2%) were anterior wall MI. More of anterior wall MI patients had cardiomegaly when compared to inferior wall MI.

In relation to ECHO, RWMA was present in 74 patients of which 47(63.5%) were anterior wall MI. More of anterior wall MI patients had RWMA when compared to inferior wall MI.

In relation to Ejection fraction , 27 patients had low EF, of which 18(66.7%) were anterior wall MI. More of anterior wall MI patients had low EF when compared to inferior wall MI.

Summary

SUMMARY

1) The incidence of anterior wall MI is more than inferior wall MI (65:35).
2) The incidence of MI is highest in the age group of 50-60 years(39%) and more in males(84%).
3) The incidence of MI is more among manual labourers(66%).
4) The incidence of MI is more among smokers(78.6%) and those with other risk factors such as obesity,hypertension,diabetes,hyperlipidemia etc.
5) Systolic and diastolic BP are higher in anterior wall MI when compared to inferior wall MI.
6) Tachyarrhythmias and other complications are more common in anterior wall MI , whereas heartblocks are more common in inferior wall MI.
7) Majority of the patients admitted in ICCU are in Class I based on Killip's classification(42%).
8) Mortality is high in anterior(72.2%) when compared to inferior wall MI.
9) Mortality is high in the age group 50-60 years(44.4%) and more in males(83.3%)
10) Mortality is high in Class IV Killip(61.1%) patients and more due to VT/VF(88.9%)

Conclusion

CONCLUSION

The overall incidence of anterior wall MI is much more than inferior wall MI. Also complications such as arrhythmias and mortality are much more in anterior wall MI than in inferior wall MI. Hence more care should be taken and close monitoring and early treatment should be instituted in all patients with anterior wall MI.

Bibliography

BIBLIOGRAPHY

1. A.R.Ness and G.Davey Smith: The Epidemiology of Ischaemic Heart Disease in *Oxford Textbook of Medicine*, David A.Warrell et al (Eds), 4th Ed, Vol 2, Chap 15.4.1.2, Pgs 912-919.
2. Luepker RV, Apple FS, Christenson RH, et al: Case definitions for acute coronary heart disease in epidemiology and clinical research studies: A statement from the AHA Council on Epidemiology and Prevention; AHA Statistics Committee; *Circulation* 2003; 108:2543
3. Curtis M.Rimmerman: Acute Myocardial Infarction in the *Cleveland Clinic Intensive Review of Internal Medicine, James K.Stoller* et al (Eds) Chap 67, Pgs 732-737.
4. Pearson TA, Blair SN, Daniels SR, et al. AHA guidelines for primary prevention of cardiovascular disease and stroke: 2002 update: consensus panel guide to comprehensive risk reduction for adult patients without coronary or other atherosclerotic vascular diseases. *Circulation* 2002;106:388–391
5. Homage to James B. Herrick:A Contemporary Look at Myocardial Infarction The 32nd Annual Herrick Lecture of the Council on Clinical Cardiologyof the American Heart Association Thomas N. James, MD ; *Circulation*. 2000;101:1874-1887

6. Yusuf S, Vaz M, Pais P: Tackling the challenge of cardiovascular disease burden in developing countries. *Am Heart J* 2004; 148:1.
7. American Heart Association : Heart Disease and Stroke Statistics—2007 Update. *Circulation* 2007; 115:69.
8. Goldberg RJ, Glatfelter K, Burbank-Schmidt E, et al: Trends in community mortality due to coronary heart disease. *Am Heart J* 2006; 151:501.
9. Braunwald E, Antman EM: Evidence-based coronary care. *Ann Intern Med* 1997; 126:551.
10. Marshall T: Evaluating national guidelines for prevention of cardiovascular disease in primary care. *J Eval Clin Pract* 2005; 11:452.
11. Boersma E, Mercado N, Poldermans D, et al: Acute myocardial infarction. *Lancet* 2003; 361:847.
12. Lopez AD, Mathers CD, Ezzati M, Jamison DT, Murray CJ. Global and regional burden of disease and risk factors, 2001: systematic analysis of population health data. *Lancet* 2006;367:1747–1757
13. Goldberg RJ, Yarzebski J, Lessard D, Gore JM. A two-decades (1975 to 1995) long experience in the incidence, in-hospital and long-term case-fatality rates of acute myocardial infarction: a community-wide perspective. *J Am Coll Cardiol* 1999;33:1533–1539.

14. Richard Drake ,Wayne Vogl- Gray's anatomy for students – 2004, P-154-181

15. Zahger D, Hod H, Gottlieb S, et al: Influence of the new definition of acute myocardial infarction on coronary care unit admission, discharge diagnosis, management and outcome in patients with non-ST elevation acute coronary syndromes: A national survey. *Int J Cardiol* 2006; 106:164.

16. Alpert JS, et al. Definition of myocardial infarction—a global consensus document of The Joint ESC/ACC/AHA/WHF/WHO Task Force, 2007

17. Marshall T: Evaluating national guidelines for prevention of cardiovascular disease in primary care. *J Eval Clin Pract* 2005; 11:452.

18. Kher N, Marsh JD: Pathobiology of atherosclerosis—a brief review. *Semin Thromb Hemost* 2004; 30:665

19. Wasserman EJ, Shipley NM: Atherothrombosis in acute coronary syndromes: Mechanisms, markers, and mediators of vulnerability. *Mt Sinai J Med* 2006; 73:431

20. Peter Libby et al: The Vascular Biology of Atherosclerosis in *Braunwald's Heart Disease – A Textbook of Cardiovascular Medicine*. Douglas P.Zipes et al (Eds), 7th Ed, Part V, Chap 35, Pgs 921-935.

21. Robert S.Schwartz et al: Pathogenesis of Atherosclerosis in *Mayo Clinic Cardiology Review*, Joseph G.Murphy (Ed), 2nd Ed, Chap 9, Pgs 113-126.

22. Peter Libby: Prevention and Treatment of Atherosclerosis in *Harrison's Principles of Internal Medicine* 16th Ed, Vol 2, Chap 225, Pgs 1430-1433.

23. Russel Ross: The Biology of Atherosclerosis in *Comprehensive Cardiovascular Medicine, Eric J.Topol* (Ed), Vol 1, Section 1, Chap 1, Pgs 14-23.

24. Cotran et al: Blood Vessels in *Robbins Pathologic Basis of Disease* 5th Ed, Chap 12, Pgs 565-567.

25. McMillian DE. Blood flow and the localization of atherosclerotic plaques. *Stroke* 1985;16:582–587

26. Malek AM, Alper SL, Izumo S: Hemodynamic shear stress and its role in atherosclerosis. *JAMA* 1999; 282:2035.

27. Berliner JA, Navab M, Fogelman AM, et al. Atherosclerosis: basic mechanisms. Oxidation,inflammation, and genetics. *Circulation* 1995;91:2488–2496.

28. Rosenberg RD, Aird WC: Vascular-bed–specific hemostasis and hypercoagulable states. *N Engl J Med* 1999; 340:1555.

29. Libby P. Inflammation in atherosclerosis [review]. *Nature* 2002;420:868–874.

30. Hansson GK. Inflammation, atherosclerosis, and coronary artery disease. *N Engl J Med* 2005;352:1685–1695.

31. Ross R, Glomset JA. Atherosclerosis and the arterial smooth muscle cell: proliferation of smooth muscle is a key event in the genesis of the lesions of atherosclerosis. *Science* 1973;180:1332–1339.

32. Ross R. The pathogenesis of atherosclerosis—an update. *N Engl J Med* 1986;314:488–500.

33. Ross R. The pathogenesis of atherosclerosis: a perspective for the 1990s. *Nature*1993;362:801–809

34. Ross R. Atherosclerosis—an inflammatory disease. *N Engl J Med* 1999;340:115–126.

35. Bruce F Waller: Nonatherosclerotic Coronary Heart Disease in *Hurst's The Heart* 11th Ed, Vol 1, Part 6, Chap 47, Pg 1174.

36. Baroldi G. Diseases of the coronary arteries. In: Silver MD, ed. *Cardiovascular Pathology*. New York: Churchill Livingstone, 1983:341.

37. Waller BF. Atherosclerotic and nonatherosclerotic coronary artery factors in acute myocardial infarction. In: Pepine CJ, ed. *Acute Myocardial Infarction*. Philadelphia: FA Davis, 1989:29–104.

38. Eliot RS, Baroldi G. Necropsy studies in myocardial infarction with minimal or no coronary luminal reduction due to atherosclerosis. *Circulation* 1974;49:1127–1131.

39. Cheitlin MD, McAllister HA, deCastro CM. Myocardial infarction without atherosclerosis. *JAMA* 1975;231:951–959.

40. Baim DS, Harrison DC. Nonatherosclerotic coronary heart disease (including coronary artery spasm). In: Husrt JW, et al (eds). *The Heart,* 5th ed. New York: McGraw-Hill, 1982:1158–1170.

41. Virmani R, Forman MB, eds. *Nonatherosclerotic Ischemic Heart Disease*. New York: Raven Press, 1989:1–428.

42. Cheitlin MD, Virmani R. Myocardial infarction in the absence of coronary atherosclerotic disease. In: Virmani R, Forman MB, eds. *Nonatherosclerotic Ischemic Heart Disease*. New York: Raven Press, 1989:1–30.

43. N.A.Boon, K.A.A.Fox: Cardiovascular Disease in *Davidson's Principles and Practices of Medicine* 19th Ed, Chap 12, Pgs 422-423,440-441.

44. Yusuf S, Hawken S, Ounpuu S, et al: Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): Case-control study. *Lancet* 2004; 364:937.

45. Lloyd-Jones DM, Leip EP, Larson MG, et al. Prediction of lifetime risk for cardiovascular disease by risk factor burden at 50 years of age. *Circulation* 2006;113:791–798. [PMID: 16461820]

46. Chuichi Kawai and Yasuyuki Nakamura: Coronary Artery Disease-*Natural History in Cardiovascular Medicine, James T.Willerson* et al (Eds), 2nd Ed, Chap 33, Pgs 691-705

47. A.J.Camm: Cardiovascular Medicine in *Kumar and Clark's Clinical Medicine* 5th Ed,Chap 13, Pgs 767-768

48. Pearson TA, Blair SN, Daniels SR, et al. AHA guidelines for primary prevention of cardiovascular disease and stroke: 2002 update: consensus panel guide to comprehensive risk reduction for adult patients without coronary or other atherosclerotic vascular diseases. *Circulation* 2002;106:388–391.

49. Greenland P, Smith SC Jr, Grundy SM. Improving coronary heart disease risk assessment in asymptomatic people: role of traditional risk factors and noninvasive cardiovascular tests. *Circulation* 2001;104:1863–1867.

50. Burke AP, Farb A, Pestaner J, et al. Traditional risk factors and the incidence of sudden coronary death with and without coronary thrombosis in blacks. *Circulation* 2002;105(4):419–424.

51. Shah PK. Thrombogenic risk factors for atherothrombosis. *Rev Cardiovasc Med* 2006;7(1):10–16.

52. BD Hoit, EA Gilpin, Myocardial infarction in young patients: an analysis by age subsets Circulation,1986, Vol 74, 712-721,

53. Prashant Joshi; Shofiqul Islam, Risk Factors for Early Myocardial Infarction in South Asians Compared With Individuals in Other Countries *JAMA. 2007*;297(3):286-294.

54. Smith SC Jr, Allen J, Blair SN, et al. AHA/ACC guidelines for secondary prevention for patients with coronary and other atherosclerotic vascular disease: 2006 update: endorsed by the National Heart, Lung, and Blood Institute. *Circulation* 2006;113:2363–2372.

55. Sonia S. Anand , Shofiqul Islam , Risk factors for myocardial infarction in women and men: insights from the INTERHEART study *Eur Heart J (2008)* 29 (7): 932-940.

56. Mosca L, Appel LJ, Benjamin EJ, et al. Evidence-based guidelines for cardiovascular disease prevention in women. American Heart Association scientific statement. *Arterioscler Thromb Vasc Biol* 2004;24:e29–e50.

57. Rissanen AM. Familial aggregation of coronary heart disease in a high incidence area (North Karelia, Finland). *Br Heart J*

1979;42:294–303.

58. Williams RR, Hopkins PN, Wu LL, et al. Evaluating family history to prevent early coronary heart disease. In: Pearson TA, ed. *Primer in Preventive Cardiology*. Dallas, TX: American Heart Association, 1994:93–106.

59. He J, Vupputuri S, Allen K, et al. Passive smoking and the risk of cor disease—a meta-analysis of epidemiologic studies. *N Engl J Med* 199 926.

60. Gordon T, Kannel WB, McGee D, et al. Death and coronary attacks i giving up cigarette smoking. A report from the Framingham stu 1974;2:1345–1348.

61. Wilhelmsson C, Vedin JA, Elmfeldt D, et al. Smoking and m infarction. *Lancet* 1975;1:415–420.

62. Eva Prescott, Merete Hippe Smoking and risk of myocardial inf women and men: longitudinal population study *BMJ 1998*; 316 : 1043

63. Kannel WB. Blood pressure as a cardiovascular risk factor: preve treatment. *JAMA* 1996;275:1571–1576.

64. Collins R, MacMahon S. Blood pressure, antihypertensive drug treat the risks of stroke and of coronary heart disease. *Br Med Bull* 1994;50:

65. MacMahon S, Peto R, Cutler J *et al*. Blood pressure, stroke, and coro

disease. Lancet 1990; 335: 765–74.

66. Whelton PK. Epidemiology of hypertension. Lancet 1994;344: 101–6.
67. Trenkwalder, P. Hendricks ,Hypertension as a risk factor for cardiovascular morbidity and mortality in an elderly German population - The prospective STEPHY II Study *,European Heart Journal (1999)* 20, 1752–1756
68. Vega GL, Grundy SM. Hypoalphalipoproteinemia (low high density lipoprotein) as a risk factor for coronary heart disease. *Curr Opin Lipidol* 1996;7:209–216.
69. Brewer HB Jr. Increasing HDL cholesterol levels. *N Engl J Med* 2004;350:1491–1494.
70. Austin MA. Triglyceride, small, dense low-density lipoprotein, and the atherogenic lipoprotein phenotype. *Curr Atheroscler Rep* 2000;2:200–207.
71. Joseph L. Goldstein, William R. Hazzard,Hyperlipidemia in Coronary Heart Disease I. Lipid levels in 500 survivors of myocardial infarction J Clin Invest. 1973July;52(7):1533–1543
72. Krolewski AS, Kosinski EJ, Warram JH, et al. Magnitude and determinants of coronary artery disease in juvenile-onset, insulin-dependent diabetes mellitus. *Am J Cardiol* 1987;59:750–755.
73. Abbott RD, Donahue RP, Kannel WB, et al. The impact of diabetes on survival following myocardial infarction in men vs women. The Framingham Study. *JAMA* 1988;260:3456–3460
74. Lundberg V Stegmayr B , Diabetes as a risk factor for myocardial infarction:

population and gender perspectives. *J Intern Med. 1997* Jun;241(6):485-92.

75. Fernandez-Ortiz A, Badimon JJ, Falk E, et al. Characterization of the relative thrombogenicity of atherosclerotic plaque components: implications for consequences of plaque rupture. *J Am Coll Cardiol* 1994;23(7):1562–1569.
76. Toschi V, Gallo R, Lettino M, et al. Tissue factor modulates the thrombogenicity of human atherosclerotic plaques. *Circulation* 1997;95(3):594–599.
77. Danesh J, Collins R, Peto R. Lipoprotein(a) and coronary heart disease. Meta-analysis of prospective studies. *Circulation* 2000;102:1082–1085
78. Haskell W. Sedentary lifestyle as a risk factor for coronary heart disease. In: Pearson T, ed. *Primer in Preventive Cardiology*. Dallas, TX: American Heart Association, 1994:173–187.
79. Poirier P, Giles TD, Bray GA, et al. Obesity and cardiovascular disease: pathophysiology, evaluation, and effect of weight loss: an update of the 1997 American Heart Association Scientific Statement on Obesity and Heart Disease from the Obesity Committee of the Council on Nutrition, Physical Activity, and Metabolism. *Circulation* 2006;113:898–918.
80. Carl J. Lavie,, Richard V. Milani Obesity and Cardiovascular Disease Risk Factor, Paradox, and Impact of Weight Loss , *J Am Coll Cardiol, 2009*; 53:1925-1932

81. Mangoni AA, Jackson SH. Homocysteine and cardiovascular disease: current evidence and future prospects. *Am J Med* 2002;112:556–565

82. Kinlay S, Ganz P. Role of endothelial dysfunction in coronary artery disease and implications for therapy. *Am J Cardiol* 1997;80:111–161.

83. Ridker PM, Cannon CP, Morrow D, et al. C-reactive protein levels and outcomes after statin therapy. *N Engl J Med* 2005;352:20–28.

84. Allan S.Jaffe et al: Diagnosis of Acute Coronary Syndromes including Acute Myocardial Infarction in *Cardiology, Micheal H.Crawford* (Ed) 2nd Ed, Section 2, Chap 12, Pgs 320

85. Jaffe AS, Babuin L, Apple FS: Biomarkers in acute cardiac disease: The present and the future. *J Am Coll Cardiol* 2006; 48:1.

86. Morrow DA, Cannon CP, Jesse RL, et al: National Academy of Clinical Biochemistry Laboratory Medicine Practice Guidelines: Clinical characteristics and utilization of biochemical markers in acute coronary syndromes. *Circulation* 2007; 115:e356

87. *Chou: Electrocardiography* in Clinical Practice 4th Ed, Chap 9, Pgs 121-141.

88. Galen S.Wagner: *Marriott's Practical Electrocardiography*, 10th Ed, Chap 7-10, Pgs 140-198.

89. *Colin Schamroth: An Introduction to ECG* 7th Ed, Section 2, Chap 11, Pgs 131-156.

90. Zimetbaum PJ, Josephson ME: Use of the electrocardiogram in acute

myocardial infarction. *N Engl J Med* 2003; 348:933.

91. Cheitlin MD, Armstrong WF, Aurigemma GP, et al: ACC/AHA/ASE 2003 guideline update for the clinical application of echocardiography: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/ASE Committee to Update the 1997 Guidelines for the Clinical Application of Echocardiography). American College of Cardiology web site, 2006.
92. Antman EM. Rutherford JD: Coronary Care Medicine: A Practical Approach. Boston, Martinus Nijhoff, 1986; and Dreifus LS, Fisch C, Griffin JC, et al:. *J Am Coll Cardiol* 18:1, 1991
93. Muhammad Bilal Siddique, Imran Fazal, Frequencies and patterns of arrythmias in anterior and inferior myocardial infarction, Pakistan Armed Forces Medical Journal, December 2009
94. Birnbaum Y, Chamoun AJ, Conti VR, Uretsky BF: Mitral regurgitation following acute myocardial infarction. *Coron Artery Dis* 2002; 13:337-344.
95. Birnbaum Y, Fishbein MC, Blanche C, Siegel RJ: Ventricular septal rupture after acute myocardial infarction. *N Engl J Med* 2002; 347:1426-1432.
96. Birnbaum Y, Chamoun AJ, Anzuini A, Lick SD, Ahmad M, Uretsky BF: Ventricular free wall rupture following acute myocardial infarction. *Coron Artery Dis* 2003; 14:463-470.
97. Stone PH et al and the millis study group – prognostic significance of location

and type of infarction independent adverse outcome associated with anterior location –Journal of American college of cardiology ;453,1998

98. Killip T, Kimball JT -Treatment of myocardial infarction in a coronary care unit. A two year experience with 250 patients. *Am J Cardiol.* Oct 1967,**20** (4): 457–64

99. Peter J Sharis , Christopher P Cannon,P-272, Evidence based cardiology 2003

Appendix

CONSENT FORM

DEPARTMENT OF GENERAL MEDICINE ,

COIMBATORE MEDICAL COLLEGE HOSPITAL.

COMPARATIVE STUDY OF ANTERIOR AND INFERIOR WALL MYOCARDIAL INFARCTION

Informed consent form for prospective participants

Principal Investigator: Dr Himal Raj.M , Junior Resident.

Research Guide: Prof. Dr S.Usha.MD. Chief, Medical Unit – III

Organization: Department of Medicine, Coimbatore Medical College Hospital.

This informed consent form has two parts

PART – I INFORMATION SHEET(to share the information about the research with you)

PART – II CERTIFICATE OF CONSENT (for signatures if you agree to take part)

(You will be given a copy of the full informed consent form.)

PART – I INFORMATION SHEET

I , Dr Himal Raj.M., Junior resident in Dept of Medicine invites you to join as participant in my research on Myocardial infarction, which is a very common problem in our country. I am going to give you information and invite you to be part of this research. You do not have to decide today whether or not you will participate in the research. Before you decide, you can talk to anyone you feel comfortable with about the research.

There may be some words that you do not understand. Please ask me to stop as we go through the information and I will take time to explain. If you have questions later, you can ask them of me, the study doctor or the staff.

Myocardial infarction is a a disease characterized by infarction and necrosis of the myocardium secondary to occlusion of the coronary blood vessels.It can involve either anterior wall or inferior wall.It could be fatal if left untreated . We are doing this research to compare the incidence ,outcome and prognosis of anterior and inferior wall myocardial infarction and thereby focus our prevention and treatment efforts in a better way.

In this study you will have to answer questions regarding your illness, undergo a physical examination , give urine and blood for tests, undergo a radiological exam of chest, an electrocardiogram and an echocardiogram.

Your participation in this research is entirely voluntary. It is your choice whether to participate or not. Whether you choose to participate or not, all the services you receive at this clinic will continue and nothing will change. You may change your mind later and stop participating even if you agreed earlier.

You will have to give details regarding your age, duration of disease, family history of the disease, any symptoms you are having at present, your past medical problems, surgeries and current medications. A doctor will examine you to look for any problems.. All the data will be recorded in a proforma. Ten ml of blood will be drawn for doing various laboratory tests to know about the status of your disease. Any excess sample will be destroyed immediately after the laboratory tests are completed. Taking the blood sample will produce some pain and there may be slight redness at the site of puncture for a day or two. Also you will have to provide 3 ml or urine for tests to detect protein. You will be subjected to a radiological exam of the chest, an electrocardiogram(ECG) and an echocardiogram will be recorded all of which are painless procedures.

On the first day you will be asked about your problems, a doctor will check you up and an ECG will be taken. You will also have to give the blood and urine samples. Subsequently, chest X ray and Echocardiogram will also be taken.

If you participate in this research you will be having a thorough check up, which may reveal some unidentified problems in you. We will promptly start the treatment for them. Also by participating you are providing valuable data that will help doctors understand this disease better and ultimately serve the patients in a better way.

We will not be providing any money for participating in this research, you may incur more expense since you will have to visit the hospital more frequently.

It is possible that if others in the community are aware that you are participating in this research, they may ask you questions. We will not be sharing the identity of those participating in the research with anyone. The information that we collect from this research project will be kept confidential. Information about you that will be collected during the research will not be identified by your name but by a number. Only the researchers will know what your number is and they will lock that information up with a lock and key. It will not be shared with or given to anyone except my research guide.

The knowledge that we get from doing this research will be shared with you before it is made widely available to the public. Confidential information will not be shared. There will be small meetings in the community and these will be announced. After these meetings, we will

publish the results in order that other interested people may learn from our research

You do not have to take part in this research if you do not wish to do so. You may also stop participating in the research at any time you choose. It is your choice and all of your rights will still be respected.

If you have any questions you may ask them now or later, even after the study has started. If you wish to ask questions later, you may contact

Dr Himal Raj M,
Junior Resident,
Dept Of Medicine,
Coimbatore Medical College Hospital
Coimbatore – 18.
Phone – 9894295330

Prof .Dr. S.Usha.MD.
Chief, Medical Unit – III,
Dept Of Medicine,
Coimbatore Medical College Hospital,
Coimbatore- 18.
Phone – 9865980015

This proposal has been reviewed and approved by the Ethics Committee of Coimbatore Medical College Hospital which is a committee whose task it is to make sure that research participants are protected from harm.

PART – II CERTIFICATE OF CONSENT

I have been invited to participate in research on myocardial infarction. I understand that it will involve answering a detailed questionnaire, undergoing a thorough physical exam, giving blood and urine samples ,an ECG,CXR and an ECHO. I have been informed that the risks are minimal and may include only slight pain and redness at sight of needle prick. I am aware that there may be no benefit to me personally and that I will not be compensated monetarily. I have been provided with the name of a researcher who can be easily contacted using the number and address I was given for that person.

I have read the foregoing information or it has been read to me. I have had the opportunity to ask questions about it and any questions I have asked have been answered to my satisfaction. I consent voluntarily to participate as a participant in this research and understand that I have the right to withdraw from the research at anytime without in anyway affecting my medical care.

Name of the participant:

_____

Signature of the participant:

_____

Date:

_____

(Day/Month/Year)

If illiterate

A literate witness must sign (if possible , this person should be selected by the participant and must have no connection to the research team)

I have witnessed the accurate reading of the consent form to the potential participant , translated to his mother tongue, and the individual has had opportunity to ask questions. I confirm that the individual has given consent freely.

Name of witness: _____ AND Thumb print of participant

Signature of witness: _____

Date : _____

(Day/Month/Year)

I have accurately read or witnessed the accurate reading of the consent form to the potential participant, and the individual has had the opportunity to ask questions. I confirm that the individual has given consent freely.

Name of the researcher:

_____

Signature of the researcher:

_____

Date: _____

(Day/Month/Year)

PROFORMA

NAME : **AGE :**

SEX: M/F

IP NO: **DOA:** **DOD:**

OCCUPATION:

DIAGNOSIS:

WINDOW PERIOD:

PRESENTING COMPLAINTS :

CHEST PAIN:

DURATION:

LOCATION:

RADIATION

ASSOCIATED SYMPTOMS :

SWEATING

PALPITATION

DYSPNEA

PND,ORTHOPNEA

SYNCOPE

GIDDINESS

NAUSEA & VOMITING

HISTORY OF PRESENT ILLNESS :

PAST HISTORY : SHT / DM / COPD

SMOKING:

ALCOHOL CONSUMPTION:

DIET: VEG/NON VEG/MIXED

MENSTRUAL HISTORY:

FAMILY HISTORY: IHD/OBESITY/DM/SHT/HYPERLIPIDEMIA

TREATMENT HISTORY: DRUG INTAKE

<u>GENERAL EXAMINATION</u>:

DYSPNEA/PALLOR/JAUNDICE/CYANOSIS/CLUBBING/PEDAL EDEMA

XANTHALESMA / TENDON XANTHOMAS/ ARCUS SENILIS

THYROID

PULSE:

BLOOD PRESSURE : HEIGHT: WEIGHT: BMI:

RESPIRATORY RATE: WAIST CIRCUMFERENCE:

TEMPERATURE: WAIST HIP RATIO:

<u>SYSTEM EXAMINATION</u>

CARDIOVASCULAR SYSTEM

APICAL IMPULSE: ***S1:*** ***S2:*** ***S3 :***

MURMUR: ***ADDED SOUNDS:*** ***PERICARDIAL RUB:***

PERIPHERAL VASCULAR SYSTEM:

RESPIRATORY SYSTEM

BREATH SOUNDS:

ADVENTITIOUS SOUNDS:

ABDOMINAL EXAMINATION

ORGANOMEGALY:

FREE FLUID:

NERVOUS SYSTEM

ANY FOCAL NEUROLOGICAL DEFICITS:

FUNDUS:

<u>INVESTIGATIONS</u>

Hb: ***TC:*** ***DC:*** ***ESR:***

RBS:

BLOOD UREA: ***SERUM CREATININE:***

S.ELECTROLYTES:

LIPID PROFILE: ***S.CHOLESTEROL:***
S.TRIGLYCERIDES:

URINE ROUTINE:

ECG:

AT THE TIME OF ADMISSION:

PRESENCE OF ARRHYTHMIAS

LOCATION OF MYOCARDIAL INFARCTION:

***AFTER THROMBOLYSIS*:**

CXR (PA VIEW):

ECHOCARDIOGRAM

Master Chart

Sno	IPNo	AGE	SEX	OccSit	SHTDM	Smoking	BMI	sBP (mmHg)	dBP (mmHg)	TC	TG	HDL	Kilip	Arrhythmia	Heart block	other comp	Death	CXR	ECHO	EF (%)
1	22880	54	M	2	APP	P	29.3	146	96	226	176	36	1	-	-	-	-	N	R	60
2	23692	62	M	3	APP	A	26.8	150	94	230	166	38	1	-	-	-	-	N	R	64
3	24471	42	M	3	AAP	P	23.8	154	92	240	174	46	2	3	-	-	-	N	R	58
4	25136	53	M	2	IPP	P	23.7	90	66	220	170	32	2	-	3	-	-	N	R	60
5	25795	49	M	3	AAA	A	23.5	166	110	180	136	52	1	4	-	-	-	N	R	62
6	25963	56	M	3	APP	P	23.8	170	116	240	170	32	2	1	-	-	-	N	R	60
7	26801	45	M	3	AAA	A	28.6	144	86	176	140	56	2	4	-	-	D	N	R	64
8	27817	55	M	1	IPP	P	24.7	136	92	260	148	34	2	-	2	-	-	N	R	60
9	28037	44	M	3	APA	A	27.8	142	88	260	136	42	1	2	-	-	-	N	R	58
10	28900	72	F	3	AAA	A	29.4	150	94	240	180	34	2	-	-	1	D	C	RP	52
11	26902	39	M	3	APP	A	27.9	170	106	250	176	36	1	-	-	-	-	N	R	62
12	29198	56	M	3	APP	P	27.3	144	84	164	150	60	3	-	-	2	-	C	RL	42
13	30221	62	M	1	AAA	A	28.4	180	110	146	148	56	1	3	-	-	D	N	R	60
14	36089	43	M	2	IPP	A	24.6	124	82	230	176	34	2	-	-	4	-	N	R	58
15	40158	55	F	3	AAA	A	24.4	170	104	170	144	46	1	3	-	-	-	N	R	62
16	44746	33	M	3	IAA	A	27.9	122	82	226	136	48	2	3	-	-	-	N	R	64
17	38558	64	F	3	APP	P	26.9	166	106	174	126	42	1	-	-	-	-	N	R	60
18	39662	44	M	3	APP	A	23.7	110	92	230	170	46	4	4	-	-	D	C	RL	36
19	40766	57	M	2	IPP	A	30.7	124	78	182	166	44	2	-	-	-	-	N	R	56
20	41870	34	M	2	APP	P	24.8	170	110	240	174	42	2	4	-	-	D	N	R	62
21	42973	48	M	3	IAP	A	27.1	126	84	242	140	32	2	-	-	4	-	N	R	60
22	44077	58	M	2	AAA	A	31.1	150	88	184	130	42	2	3	-	-	-	N	R	58
23	36390	53	F	3	IPP	A	24.2	130	84	240	150	48	1	-	-	4	-	N	R	64
24	36417	26	M	3	IAA	P	24.8	92	64	180	124	50	2	-	1	-	-	N	R	58
25	36583	45	M	1	APP	P	27.3	126	78	242	150	34	4	-	3	-	-	C	RL	30
26	36642	66	M	3	IPA	A	27.4	80	60	170	166	58	4	2	-	-	D	C	RL	32
27	37038	47	M	2	AAA	P	30.2	130	94	250	180	54	3	-	-	5	-	C	RL	40
28	37226	53	M	3	IAA	P	31.1	90	64	164	136	42	3	3	-	-	-	C	RL	44
29	36747	35	M	3	APA	P	29.3	180	96	254	140	32	1	-	-	-	-	N	R	58
30	37313	46	M	1	IPP	P	29.1	130	84	162	142	50	1	-	4	-	-	N	R	62
31	37750	59	M	3	AAA	P	30.2	134	86	186	144	46	3	3	-	-	-	C	RL	40
32	37835	76	M	2	IAA	P	27.8	142	94	190	150	42	1	3	-	-	-	N	R	62
33	38080	52	M	3	APA	P	30.7	136	86	260	146	50	2	2	-	-	-	N	R	64
34	38097	44	F	3	IPP	A	25	122	82	190	142	56	1	3	-	-	-	N	R	60
35	38510	54	M	2	AAA	P	27.1	186	110	164	144	46	2	3	-	-	-	N	R	62
36	38731	48	M	3	IAA	P	30.7	124	82	170	150	44	1	3	-	-	-	N	R	50
37	39129	68	F	3	APP	A	25	128	74	180	138	50	3	-	-	-	-	C	RL	36
38	40065	59	M	3	APA	P	27.1	116	76	182	130	58	4	-	-	-	D	C	RL	32
39	41303	37	M	3	IAA	A	26	94	66	232	180	32	1	3	-	-	-	N	R	58
40	41310	52	M	3	IPA	P	27.1	86	60	170	144	56	4	-	1	4	D	C	RL	30
41	41288	24	M	3	APP	P	30.7	126	92	160	146	44	1	-	-	-	-	N	R	60
42	41484	55	M	1	AAA	A	27.2	130	82	174	156	46	2	4	-	-	-	N	R	62
43	41832	79	F	3	APP	A	32	142	94	180	150	42	1	-	5	-	-	N	R	64
44	42121	51	M	2	AAA	P	24.4	134	84	240	184	58	2	3	-	-	-	N	R	60
45	42169	53	M	3	APP	P	31.5	174	106	174	130	50	1	-	-	-	-	N	R	66
46	42419	49	M	3	AAA	A	29.9	132	82	160	136	48	1	3	-	-	-	N	R	64
47	39313	69	M	2	IAA	P	28.8	92	66	166	162	44	3	-	3	-	-	C	RL	38
48	39537	55	F	3	AAA	A	31.5	136	84	244	132	33	2	3	-	-	-	N	R	58

49	39608	63	F	2	A	P	A	A	24.7	150	92	160	142	42	1	-	-	-	-	N	R	60
50	39755	54	M	3	A	A	A	P	22.9	156	94	250	180	32	2	-	5	-	-	N	R	56
51	39761	43	M	3	I	P	P	P	28.7	100	66	224	174	36	1	-	-	-	-	N	R	58
52	40152	55	M	3	A	P	P	P	28	146	88	230	172	34	2	3	-	-	-	N	R	62
53	40225	48	M	3	A	P	A	P	28.8	180	110	256	180	32	2	-	-	5	-	N	R	60
54	40528	84	M	1	A	A	A	P	22.6	150	94	170	130	42	1	-	4	-	D	N	R	58
55	40792	45	M	3	A	P	P	A	26.1	146	96	240	174	32	2	4	-	-	-	N	R	62
56	40844	54	M	2	I	P	A	P	25	136	82	250	184	44	2	-	-	4	-	N	R	56
57	35379	66	F	3	A	P	A	A	30.3	150	96	166	130	42	1	2	-	-	D	N	R	64
58	35395	47	M	3	A	A	P	P	23.2	170	106	180	136	50	1	-	5	-	-	N	R	66
59	35680	54	M	3	I	A	P	P	25	90	66	244	180	30	3	-	4	-	-	C	RL	38
60	35518	46	M	3	A	P	A	A	27.9	174	108	164	130	58	2	-	-	-	-	N	R	60
61	35423	69	M	1	I	P	A	P	29.1	146	96	180	140	56	1	3	-	-	-	N	R	64
62	38476	52	M	3	A	A	A	A	24.6	166	94	240	182	34	2	3	-	-	-	N	R	66
63	38697	48	M	3	I	A	A	A	26.3	130	82	162	150	42	1	-	-	-	-	N	R	64
64	39095	55	M	2	I	A	A	A	25.8	134	84	188	144	58	1	3	-	-	-	N	R	58
65	40031	33	M	3	A	A	P	P	25.1	150	88	230	180	36	2	3	-	-	-	N	R	56
66	41269	73	F	3	A	P	A	A	23.8	126	78	226	174	44	4	-	-	-	-	C	RL	40
67	41276	28	M	3	A	P	A	P	25.5	180	110	192	126	46	1	4	-	-	-	N	R	58
68	41254	58	M	3	A	A	A	P	26	110	82	170	144	66	4	-	-	-	D	C	RL	36
69	41450	51	F	2	I	P	A	A	27.4	90	66	190	130	48	2	-	2	-	-	N	R	62
70	41798	42	M	3	A	A	A	P	30.2	170	96	226	172	44	1	-	-	-	-	N	R	60
71	42087	53	M	3	A	P	A	P	25	124	74	186	140	42	4	2	-	-	D	C	RL	34
72	42135	65	M	1	I	P	P	P	26.1	92	70	240	144	32	2	3	-	-	-	N	R	60
73	42385	55	F	2	A	A	P	A	29.1	130	82	244	130	36	3	4	-	-	-	C	RL	36
74	39279	44	M	2	A	P	P	P	25.5	144	92	168	140	44	1	-	-	-	-	N	R	58
75	39503	52	F	3	A	A	A	A	24.7	126	84	170	144	46	4	3	-	-	D	C	RL	30
76	39574	45	M	3	A	A	A	P	23.3	150	98	196	144	44	1	-	-	1	-	C	RP	48
77	39608	55	M	3	I	P	A	A	26.9	136	82	180	146	42	1	3	-	-	-	N	R	62
78	39721	74	F	3	A	A	A	A	23.4	176	106	160	134	50	2	1	-	-	-	N	R	60
79	39727	47	M	3	A	P	A	P	27.9	130	94	164	126	56	1	3	-	-	-	N	R	56
80	40118	55	M	1	I	P	A	P	27.1	94	68	236	180	34	3	-	3	-	D	C	RL	32
81	40191	34	M	3	A	A	P	A	24.1	126	82	166	130	42	4	3	-	-	-	C	RL	34
82	35484	53	M	2	A	P	A	P	27.1	124	82	238	176	54	3	4	-	-	-	C	RL	36
83	35389	63	M	2	A	A	A	P	31.5	146	96	170	130	58	1	3	-	-	D	N	R	60
84	36265	27	M	3	A	P	A	P	28.3	180	110	236	136	56	2	4	-	-	-	N	R	56
85	34468	53	M	3	I	A	A	A	27.3	126	82	194	134	42	1	3	-	-	-	N	R	66
86	34662	39	M	2	I	A	P	P	29.3	150	88	192	126	60	1	-	1	4	-	N	R	62
87	34730	49	M	2	A	P	A	P	25.1	100	70	180	146	44	4	-	-	-	D	C	RL	32
88	34737	76	F	3	I	P	P	A	27.2	138	88	160	130	54	1	-	-	4	-	N	R	60
89	35018	48	M	3	I	P	A	P	29.3	136	88	233	170	42	1	3	-	-	-	N	R	56
90	35040	56	M	3	A	A	P	P	31.5	148	94	166	130	50	2	4	-	-	-	N	R	62
91	35142	38	M	3	A	P	A	P	25.6	128	82	227	136	52	1	-	5	-	-	N	R	64
92	42450	62	M	3	I	A	A	P	28.4	80	60	164	140	44	4	2	-	-	-	C	RL	30
93	42474	55	M	3	A	A	A	P	25.7	180	110	224	138	32	1	3	-	-	-	N	R	56
94	42593	87	M	3	I	A	A	A	24.7	130	82	170	150	48	2	4	-	-	D	N	R	58
95	42635	42	M	2	A	A	A	A	27.1	126	84	226	140	58	1	-	-	-	-	N	R	64
96	42855	59	M	1	A	A	A	P	27.7	150	96	162	144	50	2	3	-	3	-	N	R	62
97	41434	54	M	2	I	A	A	P	30.7	130	76	180	164	60	2	-	3	-	D	N	R	60
98	35142	68	M	3	A	P	A	P	22.6	170	100	224	142	46	1	-	-	1	-	C	RP	40
99	35920	44	M	3	I	A	A	P	25.7	90	66	246	180	30	3	-	4	-	-	C	RL	36
100	36265	58	M	2	A	A	A	P	27.6	130	94	250	176	32	2	-	-	2	-	N	R	60

ABBREVIATIONS

1ºAV Block – First degree Atrioventricular Block

2º AV Block – Second degree Atrioventricular Block

AHA – American Heart Association

AMI – Acute Myocardial infarction

AW – Anterior Wall

BMI – Body Mass Index

CHD – Coronary Heart Disease

CRP – C Reactive protein

CKMB – Creatine kinase MB fraction

CXR – Chest X Ray

DBP – Diastolic Blood Pressure

DM – Diabetes Mellitus

ECG – Electrocardiogram

ECHO – Echocardiogram

EF – Ejection Fraction

ESR – Erythrocyte sedimentation rate

HDL – High Density Lipoprotein

HOCM – Hypertrophic Obstructive Cardiomyopathy

ICCU – Intensive Coronary Care Unit

IW – Inferior Wall

LAD – Left Anterior Descending artery

LBBB – Left Bundle Branch Block

LCA – Left Coronary Artery

LVF – Left Ventricular Failure

MI – Myocardial infarction

MR – Mitral Regurgitation

NQMI – Non Q wave Myocardial infarction

NSTEMI – Non ST segment Elevation Myocardial infarction

PCI – Percutaneous Coronary Intervention

PMD – Papillary Muscle Dysfunction

QwMI – Q wave Myocardial infarction

RBBB – Right Bundle Branch Block

RCA – Right Coronary Artery

RV – Right Ventricle

RWMA – Regional Wall Motion Abnormality

SBP – Systolic Blood Pressure

SHT – Systemic Hypertension

SLE – Systemic Lupus Erythematosis

STEMI – ST segment Elevation Myocardial infarction

VT –Ventricular Tachycardia

VF – Ventricular Fibrillation

MASTER CHART

Occupation – 1-sedentary work

2-moderate exertion

3-manual labourer

Site – A- Anterior wall MI

I-Inferior wall MI

SHT- Systemic Hypertension

P-Present A-Absent

DM – Diabetes Mellitus

P-Present A-Absent

Smoking –

P-Present A-Absent

BMI – Body Mass Index

SBP – Systolic Blood Pressure

DBP – Diastolic Blood Pressure

TC – Total Cholesterol

TG – Triglycerides

HDL- High Density Lipoprotein

Killip class – 1-Class I 2-ClassII 3-ClassIII 4-ClassIV

Arrhythmias - 1-Atrial fibrillation 2-Supraventricular tachycardia

3-Ventricular extrasystole 4- Ventricular tachycardia/fibrillation

Heart blocks - 1-1st degree heart block 2-2nd degree heart block

3-Complete heart block 4- Right bundle branch block(RBBB)

5 – Left bundle branch block(LBBB)

Other complications -

1-Papillary muscle dysfunction (PMD)& acute mitral regurgitation(MR)

2-Septal perforation 3-Ventricular aneurysm

4-RV Infarction 5-Pericarditis

Death – D –Death

CXR –Chest XRay N-Normal C-Cardiomegaly

ECHO – Echocardiogram

R – Regional Wall Motion Abnormality(RWMA)

RL- RWMA + Left Ventricular Failure(LVF)

RP – RWMA +Papillary Muscle Dysfunction(PMD)